



UNDERSTANDING DERMATOLOGY

Q & A FOR MEDICAL STUDENTS

1st edition, 2000

Osama Roshdy, M.D.

More than **500** questions with their explanations

More than **80** illustrating photos

Numerous tables

Easy-to-follow outline covering the whole course topics

Designed to help you recall and review important information



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Osama Roshdy, M.D.

Lecturer

Department of Dermatology

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لا يشكر الله من لا يشكر الناس

بعد حمد الله و الشناء عليه بما هو أهله، ينبغي أن أشكر من لولاه ما كان هذا الكتاب المتواضع، ألا وهي حكومة الكويت. فقد أتاحت لي الفرصة مرتين: الأولى حين وفرت لي الوقت اللازم لإنجاز هذا العمل ، وذلك من خلال عملي لعدة سنوات في ظروف تتيح لمن شاء أن يبدع، إذا أراد. أما الثانية فتمثلت في المكتبة الرائعة بمركز العلوم الصحية و التي تضم أكثر من ألف دورية إضافة إلى ما يربو علي ثلاثة و ثلاثين ألف مرجع، متاحة لمن أراد.

بسم الله الرحمن الرحيم

ربنا لا تؤاخذنا إن نسينا أو أخطأنا

"من الآية 286 البقرة "

"إنني رأيته أنه لا يكتب أحد كتابا في يومه إلا قال في غيره، لو غير هذا لكان أحسن، ولو زيد هذا لكان يستحسن، ولو قدم هذا لكان أفضل، ولو ترك هذا لكان أجمل، وهذا من أعظم العبر، وهو دليل علي استيلاء النقص علي جملة البشر."

" العماد الأصفهاني "

NOTICE

The data included in this booklet is derived from many text and clinical books. However, the main reference is: Freedberg IM, Eisen AZ, Wolf K, Austen, Goldsmith L, Katz S, Fitzpatrick TB. **Fitzpatrick's Dermatology in General Medicine**. 5th ed. New York, NY. McGraw-Hill International Co; 1999.

The chapter on "Sexually Transmitted Diseases" is based upon information derived mainly from: Holmes KK, Sparling PF, Mårdh P-A, Lemon S, Stamm W, Piot P, Wasserheit J: **Sexually Transmitted Diseases**. 3rd ed. New York, NY. McGraw-Hill International Co; 1999

This work is dedicated to
My family

Preface

Some 10% of consultations in general practice relates to skin. This makes it very essential to study Dermatology at the undergraduate level. However, the subject still confuses many students. One reason is the lack of enough direct exposure to diagnosis and treatment during the study course. Anyone who denies that clinical dermatology is a visual specialty can never have practiced it. Never forget: **Practice makes Perfect.** Try to see as much cases as you can !!!

Another reason is that most of the about 2000 skin diagnoses have been described many years ago in an ancient language, no longer learnt at school. Such nomenclature gives rise to confusion e.g. pityriasis rubra pilaris !

I tried to help medical students preparing for the final exam by making this booklet. I choose to make it in the Q & A format as it is now widely accepted that this Socratic method is much beneficial than the Aristotelian one used in conventional textbooks as regards information retaining. In addition, it also gives the maximal yield in oral exam.

I tried to concentrate on the common skin diseases (common in *practice* and in *exam* as well!). You might do not know that only 50 out of the 2000 dermatological diagnoses constitute 90% of cases a dermatologist may see at his clinic.

Hoping to make tough complex dermatological terms more clear and palatable, I used light yellow boxes to explain the origin of the term, so the reader can understand why it has been so called. This might help to remove some of the psychological barrier he might has been suffering from e.g. **L. pityrion, bran (= نخالة)** to indicate that the term pityriasis is derived from the Latin term “*pityrion*”, which means “bran” in modern English, and “نخالة” in Arabic, signifying the “branny” nature of the lesion!

Still hoping to make the situation more easy, you could find many boxes that contain some classifications, **of quite importance**, the **histopathological** hallmarks of certain diseases, as well as data that would be **of interest** to you.

About eighty illustrating photos representing many of the relevant topics have been inserted to make your job further easier.

Lastly, I hope this modest work will sound good for all of you!

*I appreciate your inquiries, suggestions, or criticism.
You can find me very easily, just click:
oroshdy@hotmail.com*

Acknowledgments

A great deal of credit goes to **Professor Wafa'a Ramadan, M.D.**, as well as **Professor Fatma Abdul-Ghaffar, M.D.**, Department of Dermatology, Faculty of Medicine, Tanta university.

I wish to express my deep gratitude to **Professor Husein Hasab-elnaby, M.D.**, Al-Azhar University, **Professor Amani Qolqila, M.D.**, Department of Dermatology, Tanta university, **Dr. Amr Ez-Eldeen, M.D.**, Cairo University, and **Dr Hesham Hanafy, M.D.**, Tanta university, who cared to review the initial drafts of the manuscript and provided me helpful hints.

The preparation of this work would be almost impossible without using the endless facilities of the **Health Sciences Center Library**, Faculty of Medicine, Kuwait University. The unlimited access to different sections including Circulation, Periodicals, Audiovisuals and computerized literature research, should be mentioned. Special reference is to **Mrs Raja Al-Naquib** and **Mr. Ez-El-Deen Hegazy**.

I'm really grateful to **Mohamed Badr**, a 5th year Medical Student, for helping me to upload a PDF version of that work. He is the one that literally did the whole work. I wish we can work together, soon, to update the material that had been written more than 10 years ago!

Last but not least, I am also thankful to **Mr Osama Mansour**, Al-Sabah Hospital, Kuwait, for his secretarial assistance.

Some of the clinical photographs come from the departmental collection of Asa'ad Al-Hamad Dermatology Center, Al-Sabah Hospital, Kuwait. I wish to thank all those who presented them. I am most grateful to **Dr. Qasem Al-Saleh**, Director of the Center and **Dr Ayman Hasanein**.

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بسم الله الرحمن الرحيم

"فأما الزبد فيذهب جفاء ، وأما ما ينفع الناس فيمكث في الأرض"

"الرعد - من الآية 17 "

ومن لم يصانع في أمور كثيرة	يضرّس بأنيابٍ ويوطأ بمنسم
ومن يك ذا فضل فيبخل بفضله	علي قومه يستغن عنه ويؤدم
ومن هاب أسباب المنايا ينلنه	وإن يرق أسباب السماء بسلم
ومن يجعل المعروف في غير أهله	يكن حمده ذماً عليه، ويندم
ومن لم يزد عن حوضه بسلاحه	يهدم، ومن لا يظلم الناس يظلم
ومن يغترب يحسب عدوا صديقه	ومن لا يكرّم نفسه، لا يكرّم
ومهما تكن عند امرئ من خليقة	وإن خالها تخفي علي الناس، تعلم
وإن سفاه الشيخ لا حلّم بعده	وإن الفتى بعد السفاهة يحلّم

"زهير"

BASIC KNOWLEDGE

ANATOMY

• Can you NAME the skin layers?

arranged from outside, the **THREE** layers are:

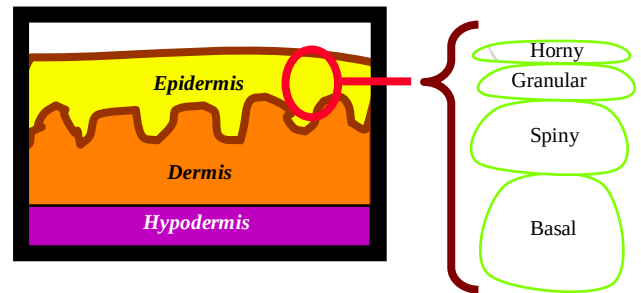
1. **EPIDERMIS**, composed of:

- ❑ Keratinocytes **main cell**

avascular, stratified squamous epithelium arranged into **4 layers** (from inside):

- **Basal** cell layer
- **Spiny** cell layer
- **Granular** cell layer
- **Horny** cell layer

- ❑ Melanocytes for **pigmentation**
- ❑ Langerhans cells for **immunity**
- ❑ Merkel cells for **sensation**



2. **DERMIS**, composed of:

- ❑ Collagen for **resilience**
- ❑ Elastin for **elasticity**
- ❑ Proteoglycans for **water maintenance**

contains:

- Epidermal appendages
 - Hair follicles
 - Sebaceous glands
 - Sweat glands (eccrine, apocrine)
- Blood vessels, nerves and lymphatics

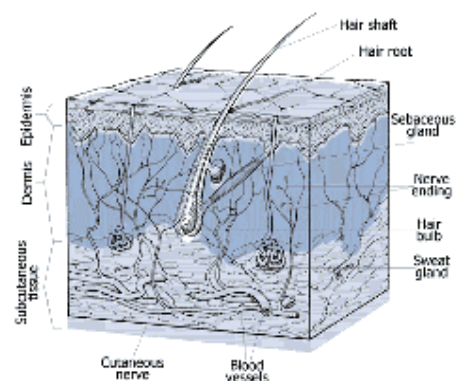
3. **HYPODERMIS**, composed of:

- ❑ Adipose tissue
- ❑ Large blood vessels and nerves

PHYSIOLOGY

• Would you mention SOME of the skin functions?

- ❑ **Mechanical support**
- ❑ **Protection**, (against invasion by: Chemicals , Particles, UV rays, Antigens, Microbes).
- ❑ **Thermoregulation**
- ❑ **Endocrine function**
 - e.g. vitamin D synthesis
- ❑ **Excretory function**
 - e.g. sweating
- ❑ **Sensation**



CLINICAL HINTS

- **How can you reach an accurate diagnosis?**

This requires

- Careful history taking
- Thorough examination
- Intelligent use of lab.

YOUR
OWN SHEET !!!!

- **What should be done before history taking?**

It is best to have a quick look on the patient (general, local) before obtaining a full history. This will prompt the right questioning.

- **How could you obtain a full history?**

ASK ABOUT :

- **the present skin condition**

- When did it start?
- Does it itch, burn or hurt?
- Where “on the body” did it start?
- How has it spread?
- How has it changed?
- What are the provocative factors?
- What is the previous treatment(s)?

What are the common dermatologic complaints?

- ✓ Pruritus
- ✓ Disfigurement

L. *prurire*, to itch

Is there a difference between itching & pruritus?

No !!

What is itching?

Peculiar irritating sensation in the skin that arouses the desire to *scratch*.

What is scratching?

Making marks on skin with nails.

- **the general health at present**

- Acute illness syndrome: fever, sweats, headache, nausea,...
- Chronic illness syndrome: malaise, anorexia, weight loss,...

- **the past history of skin disorders**

- **the past general medical history**

- Asthma, allergic rhinitis

- **the family history of skin diseases**

- *if positive*: is it inherited **OR** infective/infestive

- **the family history of other medical diseases**

- **the social and occupational history**

- Hobbies, travels abroad, relationship of rash to work or holiday, alcohol intake

- **the sexual history**

- **How can you examine the skin correctly?**

try to **find** an early “primary” lesion, to **inspect** it closely and to **define** the:

- ✓ **Type** of the individual lesion
- ✓ **Shape** of the individual lesion
- ✓ **Arrangement** of the multiple lesions
- ✓ **Distribution** of the multiple lesions

What is “**lesion**”?

Any single small area of skin disease

What is “**eruption**”?

A rapidly developing dermatosis, specially when appearing as a local manifestation of a general disease

L. *eruptio*, to break out

What is “**rash**”?

Eruption

• What are the types of skin lesions?

✓ **Primary lesions:**

early “initial” lesions **not altered** by trauma, infection, manipulation or natural regression.

✓ **Secondary lesions:**

lesions **produced** by trauma, infection, manipulation or natural regression.

• Can you define some of the **PRIMARY** lesions?

Macule: a circumscribed area of change in normal skin colour; impalpable.

Patch: same as macule, > 0.5 cm in diameter.

Papule: a superficial solid lesion, < 0.5 cm in diameter.

Plaque: a plateau-like elevation above skin surface that occupies a relatively large surface area in comparison with its height above the skin.

Nodule: palpable solid lesion that is rather **DEEP**.

Vesicle: a circumscribed, elevated superficial cavity containing fluid.

Bulla: same as vesicle, > 0.5 cm in diameter.

Pustule: a circumscribed raised lesion containing a purulent material.

Abscess: a localised accumulation of purulent material so deep in the dermis or hypodermis that pus is not visible on skin surface.

Wheal: (page 34)

Burrow: (page 26)

Comedo: (page 30)

Petechiae: (page 36)

Ecchymoses: (page 36)

Cyst: abnormal sac containing fluid or semisolid material (with a membranous lining)

Telangiectasia: visible, dilated, small, superficial blood vessel

L. *macula*, a spot
L. *papula*, a pimple
F. *plaque*, a plate
L. *vesicula*, a little bladder
L. *bulla*, a bubble
L. *pustula*, a pustule
L. *nodulus*, a small knot

• Can you define some of the **SECONDARY** lesions?

Scale: flat plate of stratum corneum, cast off from the skin

Crust: dried tissue fluid

Scab: dried blood

Lichenification: skin thickening with increased skin markings

Ulcer: focal loss of **the whole** epidermal thickness, extending into the dermis **scarring**

Erosion: focal loss of **part** of the epidermal thickness **non scarring**

Excoriation: linear (or punctate) **erosion, due to scratching**

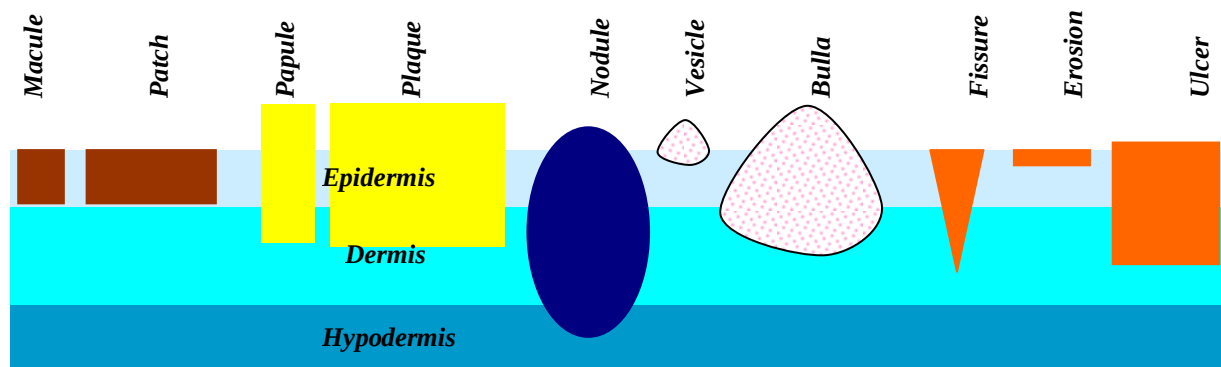
Fissure: linear crack with sharply defined walls “deeper than excoriation”

Sinus: tract leading from a suppurative cavity, to the skin surface

Scar: new connective tissue formations, replacing lost tissue

Sclerosis: hardening/induration “of the skin”

F. *escala*, a shell
L. *crusta*, a shell
L. *ulcus*, a sore
L. *erodere*, to gnaw away
L. *ex*, out of
corium, skin

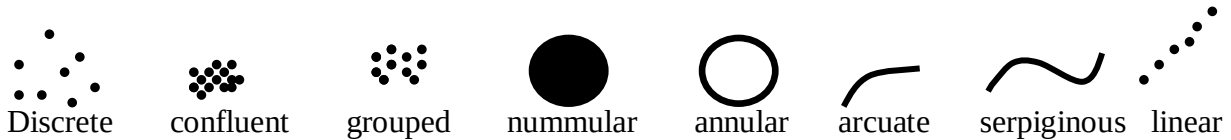


• Can you mention SOME of the lesion shapes?



dome pedunculated verrucous umbilicated flat acuminate annular arcuate linear

• Can you mention SOME of the lesions arrangements?



Discrete confluent grouped nummular annular arcuate serpiginous linear

• What do you know about the lesion distribution?

- ❑ **Extent:** local, regional, generalised, universal
- ❑ **Pattern:** symmetrical/asymmetrical
- ❑ **Characteristic location:** flexors, extensors, exposed, seborrhoeic,.....

• How do we divide skin diseases?

- ❑ **Aetiopathological** classification:
 - Inflammatory: infective, rheumatic, eczematous psoriasiform, rheumatic, etc...
 - Neoplastic: benign, malignant
- ❑ **Syndromic** classification: papulosquamous, blistering, rheumatic, pigmentary, etc...

• What are the therapeutic options in dermatology?

- ❑ Drugs
 - ✓ Topical
 - ✓ Systemic
- ❑ Surgery
 - ✓ Excision
 - ✓ Electrodesiccation
 - ✓ Cryotherapy
 - ✓ Laser
 - ✓ Peeling
 - ✓ Abrasion
 - ✓ Transplantation
- ❑ Irradiation
 - ✓ Radiotherapy
 - ✓ Phototherapy

• What is the general formula of topical preparations?

- ❑ Vehicle
- ❑ Active ingredient e.g. corticosteroids, antibiotics, antifungals,...

• What do you mean by a “vehicle”?

A base used as a container for the therapeutic modality.

It contains a mixture of powder, water and/or grease (oil) e.g.

- ❑ **Lotion** powder **in** water for weeping lesions
- ❑ **Cream** oil **in** water for weeping crusted lesions
- ❑ **Ointment** water **in** oil for dry scaly lesions
- ❑ **Paste** powder **in** oil for longstanding cases

VIRAL INFECTIONS

- Could you please, tell me SOME of the viral skin diseases?

	Disease	Organism
Maculopapular	Measles*	Measles virus
	German measles*	Rubella virus
Papulonodular	Warts	HPV
	Molluscum contagiosum	MCV; a pox virus
Vesiculobullous	Herpes simplex	HSV-1, HSV-2
	Chicken box	VZV
	Herpes zoster	VZV
AIDS		HIV

*You studied it well in paediatrics !!

WARTS

L. verruga, wart



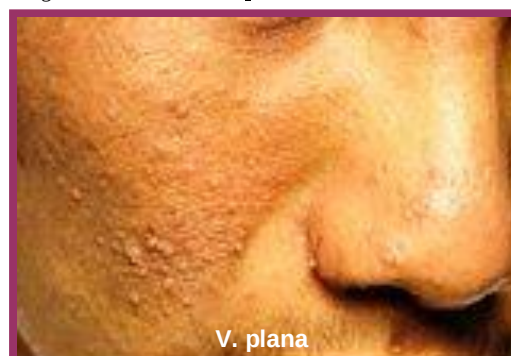
- What is the cause of warts (verrucae)?

Human papilloma virus (HPV). The virus has >70 subtypes (based on pattern of DNA content).

Infection follows **DIRECT** or indirect contact. The virus passes through minor skin abrasions.

[**Direct** contact: skin-skin. **Indirect** contact: through common objects e.g. towels]

- Are some people more susceptible?
YES. Cell mediated immunity (CMI) of the host is the critical factor.
- What is the age of highest incidence?
Around puberty.
- How long is its incubation period?
Up to 20 months.
- Can HPV live in the body in a dormant state?
YES.



- What is the clinical presentation of the main types of warts?

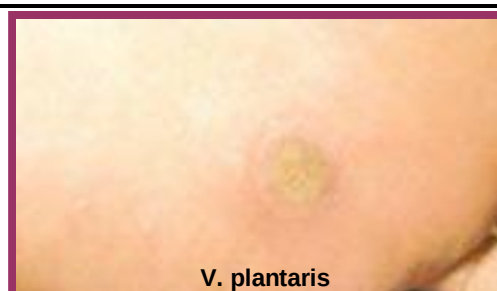
Clinical type	Site	Presentation	HPV type	Differential
Common (v. vulgaris)	Hands	Endophytic*, scaly, rough, skin-colored <u>papules</u>	2, 4	Molluscum
Plantar (v. plantaris)	Soles	Hyperkeratotic, painful <u>papule</u> / <u>plaque</u> ; studded with black dots	1	Callosity, Corn
Flat (v. plana)	Face & Hands (Children)	Small (2-5mm) flat-topped, hyper-pigmented <u>papules</u> ; multiple	3, 10	Lichen planus
Anogenital (C. acuminatum)**	Anogenital region	Moist, exophytic*, <u>papules/nodules</u> , variably sized; STD	6, 11	C. latum**

* Endophytic: infiltrative, Exophytic: cauliflower-like

** C. = condyloma, pl. condylomata

G. condyl, a knob
L. acuminatus, pointed
latus, broad

Affecting >4 millions in USA.
 50% of ♀ are asymptomatic.
Symptoms include: itching, pain,
 burning, tenderness, psychic impact.



- **Do warts spread?**

YES, especially if they are injured.

- **Shall you give some examples of painful warts?**

plantar, subungual, infected

- **Can you differentiate between *C. acuminatum* & *C. latum*?**

	<i>C. acuminatum</i>	<i>C. latum</i>
Cause	HPV	<i>T. Pallidum</i>
Morphology	Papule: Cauliflower Pedunculated	Papule: Flat Sessile
Colour	Pink	Greyish-white
Induration	Not	Indurated
Bleeding	Easy	Not
Lymphadenopathy	Absent	Generalised
Dark-ground	-ve	+ve
Serology	-ve	+ve
Treatment	See later	Antisymphilitic



- **Can warts cause cancer?**

YES. Certain types of HPV can, e.g. -**Cervical carcinoma** (HPV type 6, 11, 16)

-**Laryngeal carcinoma** (HPV type 6)

- **Could it regress spontaneously?**

YES, in some lesions within 2 years. Also, treatment of one or more lesions may lead to regression of the remaining ones.

- **What is the ideal treatment for warts?**

A viricidal agent, specifically effective against HPV.

- **Is it available?**

NOT yet, unfortunately !

What is its histopathological hallmark?

-**Hyperkeratosis**: thickening of horny layer

-**Acanthosis**: thickening of spiny layer

-**Papillomatosis**: dermal papillae extend up

- **What is the aim of the CURRENT treatment modalities?**

- ❑ Destruction of the lesion, hoping to destroy the virus.
- ❑ Immunomodulation e.g. *interferon* injection, *imiquimod* cream

- **Can you mention SOME of the available destructive methods?**

Destructive method	Wart type
Chemical	
Chloracetic acid (caustic)	Common
Salicylic acid (keratolytic)	Common, plantar
Podophyllin* (cytotoxic)	Genital
5-Fluorouracil* (cytotoxic)	Flat
Surgical	
Cryotherapy	All
Electrodesiccation	Common
Laser (CO ₂ , YAG)	Resistant
Excision	Resistant

* contraindicated in pregnancy.

- **Can you tell the patient that the lesion will “NEVER RECUR” after treatment?**

NO.

MOLLUSCUM

- **What is the cause of molluscum contagiosum?**

MCV {Molluscum contagiosum virus}; a pox virus;

L. *molluscus*, soft

- **How is it transmitted?**

By **Direct** contact.

- **What is its clinical picture?**

Single/multiple, shiny, pearly white, hemispherical, umbilicated, **PAPULE** (s).
Cheesy material can be squeezed out of the lesion.

- **Where does it preferably occur?**

- ❑ Children: face, trunk (temprates), extremities (tropics)
- ❑ Adults : genitalia (♂ > ♀)

- **What is the main differential diagnosis?**

- ❑ Single : basal cell carcinoma
- ❑ Multiple: warts.



- **Is it self-limited?**

MOSTLY, within 6 months.

- **How can you treat it?**

Via Destruction

-curettage
-cryotherapy
-electrodesiccation
-laser

What is its histopathological hallmark?
Epidermal downgrowth, containing numerous ***intracytoplasmic inclusions***.

HERPES SIMPLEX

- **What is the causative agent of herpes simplex?**

- ❑ Herpes simplex virus type 1 (HSV-1) mainly **oro-facial** mostly acquired in **childhood**
- ❑ Herpes simplex virus type 2 (HSV-2) mainly **genital** mostly acquired in **adulthood**

- **How common is it?**

World wide population studies showed positive serology in:

- ❑ HSV-1: 70-80% about one ***half*** asymptomatic
- ❑ HSV-2: 25-30% ***mostly*** asymptomatic

G. *herpo*, to creep

- **How is the virus transmitted?**

Directly, the virus passes through mucous membranes or minimal skin abrasions after close contact.

HSV is shed **from** clinical lesions as well as **from** asymptomatic carriers.

- **Could it be a droplet infection?**

NO.

- **Is it a recurrent condition?**

YES, with a variable frequency.

- **What is the meaning of “primary infection”?**

The first exposure to the virus in a seronegative patient.

- **What happens during such an episode?**

Following invasion, HSV replicates in the skin & mucous membranes.

It migrates via sensory nerves to the dorsal root ganglia to remain dormant “**latent** infection”.

- **Are primary episodes always manifest?**

NO. Most cases pass unnoticed.

- **What are the manifestations of primary episodes?**

- **History:** Constitutional symptoms

- **Exam. :** Sudden onset of grouped **VESICLES** on erythematous base, that ulcerate & crust.
Regional lymph nodes are enlarged and tender.

- ✓ Type 1: **gingivostomatitis**

may interfere with swallowing

- ✓ Type 2: **vulvovaginitis/balanitis**

may lead to urine retention

- **What happens during the recurrent attacks?**

Latent virus is **reactivated** and migrates down the sensory nerves, from dorsal ganglia to the original skin site of primary infection.



- **Can you mention some of the conditions, which may reactivate the virus?**

- Sunlight exposure

- Respiratory infections (hence the name “**cold sores**”)

- **What are the manifestations of recurrent attacks?**

- **History:** itching / tingling may precede blisters or erosions, *recurring in the same site*.

- Severe stress

- Local trauma

- **Exam. :** Initial lesion: grouped **VESICLES** on erythematous base

Secondary lesions: crusted ulcers or erosions “**more common**”

- **What are the clinical differences between primary episode and recurrent attacks?**

The **recurrent** attacks has:

- Prodromal itching / tingling
- **NO** constitutional manifestations
- Smaller, closer lesions
- Better response to treatment

How can you confirm diagnosis?

- Viral culture: (the gold standard, not easy)
- MIF: to detect viral antigens
- PCR: to detect viral DNA
- Serology: to detect viral antibodies
- Tzank smear: to detect ballooning degeneration

- **What is the differential diagnosis of herpes simplex?**

- **Oral** : aphthous stomatitis, severe oral candidiasis, pharyngitis, pemphigus vulgaris.

- **Genital:** other causes of genital ulcers (page 56)

- **Can you remember some of its complications?**

- Secondary infection

most common

- Cervical carcinoma

most serious

- Corneal ulcers/scars

- Erythema multiform

- Neonatal infection (*in mother's prenatal attack, **Caesarean** is a must*)

What is the drug of choice?

ACYCLOVIR tablets, cream, IV
Several members of the same family have been recently available e.g. valacyclovir, famciclovir

What is its mechanism of action?

Inhibits polymerase enzyme.

VARICELLA

L. varius, spotted
To signify the **polymorphic**
nature of the eruption

- **What is varicella “i.e. chickenpox”?**

This is the primary infection with varicella-zoster virus (VZV).

- **How could it be transmitted?**

- ❑ Droplet infection
- ❑ Direct contact (highly contagious)

- **Who is specially at risk?**

Children.

- **How long is the incubation period?**

1-3 weeks.

- **How long is the infectivity period?**

The infected person can infect others starting on the 4th day of infection (before any clinical lesions appear), until all lesions have crusted.

- **Shall you mention the clinical picture?**

Following a mild prodrome, successive crops of pruritic **PAPULO-VESICLES** appear.

They progress rapidly to **pustules** and crusted erosions. No scars follow.

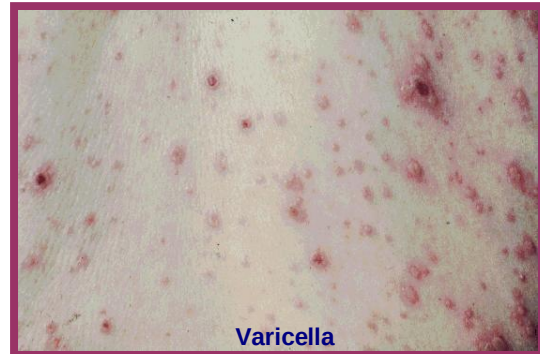
Lesions are **centripetal** i.e. most extensive on the trunk & face.

- **Do you know some of the complications?**

- ❑ Children: Secondary infection, Reye's disease.
- ❑ Adults: Pneumonia, encephalitis.

- **How would you treat it?**

- ❑ Symptomatic measures e.g. menthol, oral antihistamines, antibiotics
- ❑ Active measures (acyclovir)
- ❑ Preventive measures (vaccination)



خير الكلام

عن رَسُولِ اللَّهِ - صَلَّى اللَّهُ عَلَيْهِ وَسَلَّمَ - انه قال:

لا يقف أحدكم موقفا يظلم فيه امرؤ مسلم، فإن اللعنة تنزل علي من حضر ولم يدفع عنه

إن المرء ليلقي بالكلمة، لا يلقي لها بالا، تهوي به في النار سبعين خريفا

HERPES ZOSTER

- What is shingles “Herpes zoster”?

It is the acute skin infection due to **reactivation** of VZV (latent in the dorsal ganglia).

- What reactivates the virus?

Host immunosuppression.

L. *cingulum*, a girdle

G. *zoster*, a girdle

- How does it present?

- Prodromal pain
- Painful/pruritic, grouped **VESICLES** or crusts on erythematous base, in a **dermatomal** distribution

- How long is the course?

2-3 weeks.

- What are the most affected sites?

- Dermatomes innervated by **thoracic** nerves
- „ „ „ 1st branch of **trigiminal** nerve



H. Z.: Grouped vesicles

- Who gets zoster?

Any one who **HAD HAD** varicella, may get it.

- Who is especially at risk?

- Old-aged (>50)
- Immunocompromised e.g. AIDS



H. Z.: Dermatomal distribution

- Can it recur?

YES, usually in the same dermatome (<1%, specially in immunocompromised).

- Is it contagious?

DIRECT contact with lesions may lead to **VARICELLA** (in a susceptible host, **NEVER** exposed before to VZV)

L. *contagio*, to touch closely,
signifying transmission by contact

- Shall you mention some of its complications?

- Post-herpetic neuralgia **most common** (more in older patients)
- Scarring, secondary infection
- Keratitis, corneal ulcers
- **Ramsy-Hunt syndrome**: facial palsy following affection of genicualte ganglion.
- Meningio-Encephalitis.

- How can you treat it?

- Symptomatic measures e.g. analgesics
- Active measures (acyclovir, specially indicated for compromised)

وإذا مت لست أعدم قبراً
نفس حر ترى المذلة كفراً

أنا إن عشت لست أعدم قوتاً
همتي همة الملوك ونفسي

الإمام الشافعي

BACTERIAL INFECTIONS

- **Do microorganisms normally reside on the skin?**

YES. Normal skin has resident flora of harmless microorganisms **e.g.** *Staph. epidermidis* and *pityrosporum*.

- **What is the most important defence against bacterial invasion?**

An intact stratum corneum.

- **What is the difference between primary and secondary skin infections?**

Whether infection occurs in apparently normal (**primary**) or already diseased (**secondary**) skin.

- **Would you enumerate SOME bacteria pathogenic to skin?**

Organism	Disease
<i>C. Minutissimum</i> *	Erythrasma
<i>Strept. pyogenes</i>	Impetigo, ecthyma, cellulitis, erysipelas
<i>Staph. aureus</i>	Impetigo, folliculitis
<i>Mycobacteria</i>	TB, leprosy
<i>Spirochetes</i>	Syphilis

**Corynebacterium Minutissimum*

L. *inter*, between
tero, to rub

- **What is intertrigo?**

Dermatitis occurring between two opposing surfaces of the skin e.g. groins, axilla. It is most common in the obese and in warm humid environment. **Causes** include: dermatophytosis, moniliasis, erythrasma, streptococcal infection, seborrhoeic dermatitis, inverted psoriasis.

G. *erythro*, red

- **What is erythrasma?**

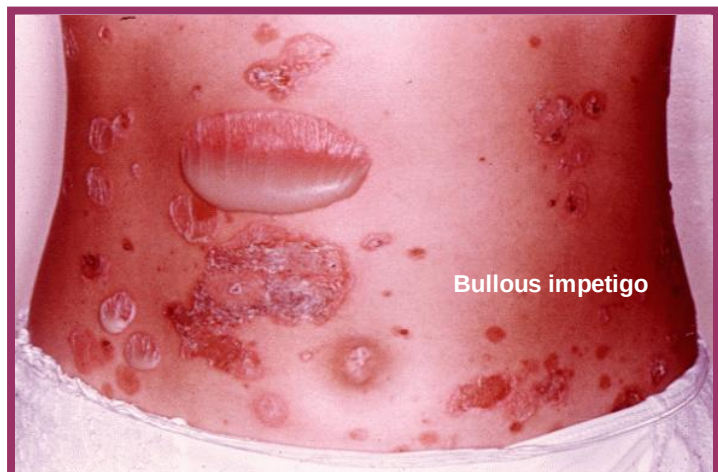
A chronic bacterial **intertrigo** caused by *Corynebacterium Minutissimum*.

- **How does erythrasma present clinically?**

Finely scaly, dry, brownish red, well demarcated **PATCHes**, mainly affecting axilla & groins.
Wood's light (p.19): coral-red fluorescence.

- **How can you treat it?**

ERYTHROMYCIN: Topical / Systemic



IMPETIGO

L. impeto, to attack

- **What is impetigo?**

A primary, superficial pyogenic skin infection, caused by either Staph. aureus or Strept. pyogenes. It is more common in **summer**, affecting mainly ♀.

How common is impetigo?
10 % of cases in Derma Clinics.

- **What are its clinical variants?**

Impetigo contagiosum

- ❑ Due to group A β -haemolytic **Streptococci** i.e. **Strept. Pyogenes** (*Staph. aureus* are secondary invaders).
- ❑ Superficial, stuck-on, honey-coloured **CRUSTS**, over **erosions**, following transient vesiculopustules.
- ❑ Mostly on the **face** (but any other site can be affected), of **pre-school** children
- ❑ Should be differentiated from: kerion and infected scabies.

Bullous impetigo

- ❑ Due to **Staphylococci** (*Staph. aureus* producing exfoliatin exotoxin leading to epidermal split)
- ❑ Thin flaccid **BLISTERS** (cloudy contents / erythematous base), mostly in **newborns**
It may collapse producing yellowish **crusts** or rupture leaving **erosions**.
- ❑ Should be differentiated from herpes simplex, and congenital syphilis.

Circinate impetigo

- ❑ Due to **either** peripheral extension of a single lesion **or** fusion of multiple lesions.
- ❑ Should be differentiated from Tinea circinata, circinate psoriasis or pityriasis rosea.

Ulcerative impetigo “ecthyma”

- ❑ Due to group A β -haemolytic **Streptococci** i.e. **Strept. Pyogenes**
- ❑ Thick adherent dirty **crust**, overlying a punched-out **ulcer**, surrounded by erythema.
Heals with **scarring**. More common on the **lower limbs** of **children & elderly**.
- ❑ Should be differentiated from leishmaniasis.

Bockhart's impetigo

- ❑ This is an **acute superficial folliculitis**.
- ❑ Small, dome-shaped pustules at the openings of hair follicles, often on children's legs

Streptococcal intertrigo*

-should be differentiated from other causes of intertrigo (see page 11)

* **NO LONGER** considered impetigo, mentioned for HISTORICAL purposes.

Furfuraceous impetigo*

-a manifestation of **ATOPY** that was thought to be a variant of impetigo.

- **Can you mention SOME of the complications of impetigo?**

- ❑ Acute glomerulonephritis **most serious**, three weeks after invasion with nephritogenic *streptococci*
- ❑ Autoinoculation **most common**

- **How shall you treat it?**

- ❑ **Curative**:
 - ✓ Topical: - Crust removal, using soap and water or potassium permanganate solution
 - Antibiotics e.g. mupirocin, bacitracin, fucidate
 - ✓ Systemic: - Antibiotics e.g. cefuroxime, penicillin, erythromycin
- ❑ **Supportive, Symptomatic**
- ❑ **Preventive**: personal hygiene

CELLULITIS

- **What is cellulitis?**
An acute infection of dermis & hypodermis, produced by *Strept. pyogenes*.
- **What is erysipelas?**
A superficial variant of **cellulitis**, with marked lymphatic involvement.
- **Can you mention the clinical picture of erysipelas?**
 - ❑ Erythematous, hot, oedematous, indurated (*peau d'orange*), sharply-demarcated **PLAQUE**.
In severe cases, overlying epidermis may show bullae, pustules or necrosis.
 - ❑ Lesions occur mostly on the **face** (adults) or **abdomen** (infants).
 - ❑ Self-limited within **1-2** weeks, with hyperpigmentation.
- **Is it recurrent?**
YES, it might.
- **How can you differentiate it from cellulitis?**
Cellulitis is characterised by:
 - ❑ Milder symptoms
 - ❑ Ill-defined border
 - ❑ Deeper location, with more infiltration and oedema
 - ❑ Possibility of abscess formation and skin necrosis
- **Is erysipelas a dangerous disease?**
YES, it might be **fatal** (thrombosis, bacteraemia, toxæmia, septicaemia)
- **How would you treat it?**
 - ❑ Curative -Systemic: antibiotics e.g. penicillin G/V, vancomycin
-Topical : cool dressing
 - ❑ Supportive, Symptomatic



تجد ذا عفة فليعلِّ لا يظلم
عن جهله وخطاب من لا يفهم
ومن الصداقة ما يضر ويؤلم!

أبو الطيب

والظلم من شيم النفوس فإن
ومن البلية عدلٌ من لا يرعوي
ومن العداوة ما ينالك نفعه

FOLLICULITIS

- What is folliculitis?
Inflammation of the hair follicle.

- Shall you, please classify it?

□ Superficial

- ✓ Acute e.g. **Bockhart's impetigo**
- ✓ Chronic e.g. **Acne vulgaris**

□ Deep

- ✓ Acute e.g. **Furuncle / Carbuncle**
- ✓ Chronic e.g. **Sycosis barbae**
Pseudo folliculitis



Bockhart's impetigo



Furuncle



Carbuncle

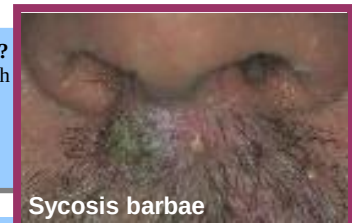
- What is the difference between furuncle and carbuncle?
 - Furuncle (Boil): affects one hair follicle.
 - Carbuncle : affects several close hair follicles
- How does furuncle present?
 - Painful, solitary (or multiple), erythematous, deep seated, follicular/perifollicular **NODULES**.
 - Mostly on areas subject to friction & perspiration e.g. neck, axilla, buttocks.
 - More common in **winter**, affecting mainly **middle** and **old** ages, especially ♂.
 - Heals with violaceous scar.

- Mention some of its complications?

- Carbuncle
- Cellulitis

How do sycosis barbae present?

- Discrete follicular **pustules**, each pierced by a hair, in the beard.
- Crustation follows, ending by scarring. New lesions develop as the old ones heal.



Sycosis barbae

- What is its differential diagnosis?

Other causes of folliculitis.

What is its differential diagnosis?

- Tinea barbae: - Loose broken-off hair
- Nodular swelling

- Why do they recur?

- **Staph.** Carriage (i.e. becoming part of the resident flora) most important
- Mechanical skin damage e.g. scratching
- Immunocompromisation
- Diabetes mellitus

- How can you treat it?

- Curative
 - ✓ Systemic:- Oral difloxacin
 - ✓ Topical :- Hot fomentation, *for early simple lesions*
- Incision & drainage, *if fluctuant*
- Supportive, Symptomatic

What is "pseudo folliculitis"?

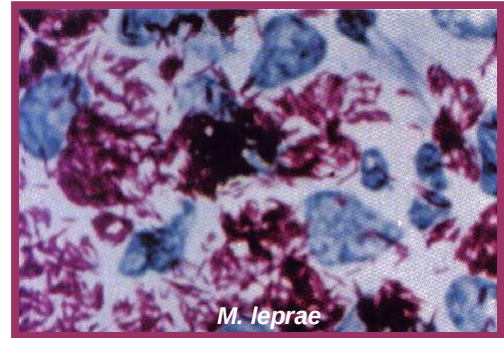
- Erythematous **papulo-pustules**, with skin-buried hair.
- It affects mainly the beard of blacks.
- It results from ingrowing hair, which predisposes to 2ry infection, caused mainly by **Staph. aureus**.



Pseudofolliculitis

MYCOBACTERIA: LEPROSY / TB

- **Would you define Mycobacteria?**
Aerobic, slowly-growing, non-spore-forming, **immotile** bacilli with high **LIPID** content in cell wall.
- **What is the meaning of “acid fast” & “alcohol fast”?**
The organism can retain the staining dye “carbolic fuchsin” after washing with acid or alcohol. This is the most useful staining characteristic of Mycobacteria.



- **Shall you mention some of the important Mycobacteria, pathogenic to humans?**
 - ❑ *M. leprae*
 - ❑ *M. tuberculosis*

- **What is leprosy (Hansen’s disease)?**
It is a granulomatous disease caused by *M. leprae* (Hansen, 1873).

How common is leprosy ?

It is a world wide disease
(about **10-12** millions cases throughout the world)

Is it an endemic disease ?

YES, in 53 countries {tropics and subtropics e.g. India (4 millions) & Egypt (Gharbia, Asiut)}.

What is the source of infection ?

Untreated open cases of **multibacillary** leprosy. Prolonged close contact is mandatory.

What is its major portal of entry?

Most probably via nasorespiratory route
(**droplet infection**).

- **Is it a systemic disease?**
YES. It has involved every organ **EXCEPT** CNS & lungs.

- **What are its preferable sites?**
 - ❑ Peripheral nerves (Schwann cell): **target cell**
 - ❑ Skin (dermis).

Are children more susceptible?

YES. Up to **60%** of the children of leprotic parents develop the disease.

Is it more common in ♂?

YES, 2:1

- **What is the fate of infection?**
After infecting the body, most cases recover naturally.
Only **susceptible** persons develop a spectrum of diseases.

- **What do you know about the SPECTRUM of leprosy?**
According to the degree of the patient’s specific CMI against *M. leprae*,

FIVE types of leprosy can be recognised, in order of ↓CMI:

- ✓ Tuberculoid (TT) **localised lesions / few bacilli**
- ✓ Borderline tuberculoid (BT)
- ✓ True borderline (BB)
- ✓ Borderline lepromatous (BL)
- ✓ Lepromatous (LL) **generalised lesions / many bacilli**

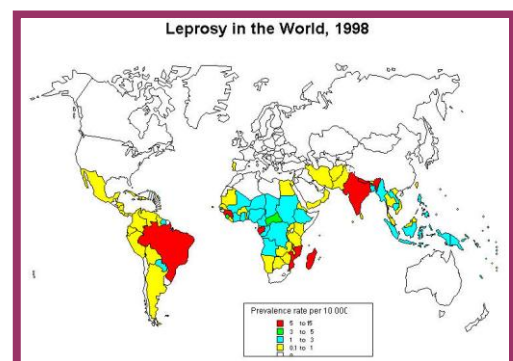
Which is the commonest?
Tuberculoid (TT)

• What is **indeterminate** leprosy?

The very early lesions following infection.
It manifests as a single, ill-defined erythematous or hypopigmented **macule**.
It may **recess** spontaneously or **progress** to other types.

• What is **pure neural** leprosy?

Cases with **neural** manifestations but **NO** skin lesions.
It is usually **mononeural**, mostly **tuberculoid**.
Sensory changes precede the motor ones.
It is **common** in Egypt.



• Can you mention the most important features of the main types of leprosy?

	TUBERCULOID	BORDER-LINE	LEPROMATOUS
Skin lesions			
Type	MACULE , plaque	PLAQUE “annular”	NODULE , plaque
Number	Single/few	Several	Very many
Size	Large	Large & small	Small
Symmetry	Asymmetrical	Symmetrical	Symmetrical
Surface	Rough, dry, scaly	Variable	Shiny, smooth
Edge	Sharp	Sharp	Vague
Sensation	Absent*	Variable	Intact, in early lesions
Hair	Lost	Moderate ↓	Not affected
Nerve lesions	Single, <u>within lesion's vicinity</u>	Several	Many, <u>peripheral</u>
Other organ lesion	----	----	Nose: crustation, epistaxis Eye: keratitis Testis: infertility
Acid-fast bacilli			
Slit-skin smear	Absent	Moderate	So many
Nasal scrap	Absent	Absent	So many
Lepromin test	+++	+	-
Course	Stable	Unstable	Stable
Prognosis	Good	Variable	Poor

*temperature is lost first

• What is lepromin test?

Intradermal injection of crude preparation of *M. lepra* into the host “to determine the hypersensitivity”. It has no value in the diagnosis. However, it is very valuable as a **PROGNOSTIC** test.

• What are the cardinal diagnostic features of leprosy?

- ❑ Cutaneous anaesthesia
- ❑ Nerve enlargement/tenderness
- ❑ Demonstration of *M. leprae* in the skin, via:
 - ✓ Slit-skin smear
 - ✓ Skin biopsy “using **FITE** stain”

• What is the difference between.....?

- ❑ **Bacteriologic index**: the number of all bacilli in oil immersion field
- ❑ **Morphologic index**: the percentage of living bacilli in ~ ~ ~

• Can you define “pauci” and “multi” bacillary?

- ❑ **Paucibacillary**: <10 bacilli at all sites (Bacterial Index of < 2).
- ❑ **Multibacillary**: 10 or more bacilli at any site (Bacterial Index of ≥ 2 or more).

إِنَّ الْهَوَانَ حِمَارُ الْقَوْمِ يَعْرِفُهُ
 وَلَنْ يَقِيمَ عَلَى خَسْفٍ يُسَامُ بِهِ
 هَذَا عَلَى الْخَسْفِ مَرْبُوطٌ بِرُمْتِهِ
 فَإِنْ أَقَمْتُمْ عَلَى ضَيْمٍ يُرَادُ بِكُمْ
 وَفِي الْبِلَادِ إِذَا مَا خِفَتْ نَائِرَةٌ
 وَالْحُرُّ يُنْكِرُهُ وَالرَّسَلَةُ الْأَجْدُ
 إِلَّا الْأَذْلَانِ عَيْرُ الْأَهْلِ وَالْوَتْدُ
 وَذَا يُشْعُجُ فَمَا يَرِثِي لَهُ أَحَدُ
 فَإِنَّ رَحْلِي لَكُمْ وَالِ وَمُعْتَمَدُ
 مَشْهُورَةٌ عَنْ وُلَاةِ السَّوِّ مُبْتَعَدُ

المتنبي

• **What are the commonest complications of leprosy?**

- ❑ **Traumatic ulcers** and bone damage, in anaesthetic limbs (TT)
- ❑ Reactional states (BT, BB, BL)
- ❑ Saddle nose, ichthyosis, testicular atrophy (LL)

• **Is it a fatal disease?**

YES, LL can produce death, due to:

-Pulmonary TB
-Amyloidosis (2ry)
-Nephritis
-Severe reactions

• **What are the reactional states of leprosy?**

Two types of reactions may occur during the course of the disease:

	Type 1 "reversal / lepra"	Type 2 "Erythema nodosum leprosum"
Type involved	BT, BB, BL	BL, LL
Mechanism	cell mediated	immune complex
Clinically	exaggerated original lesions	new lesions
Prodrome	-ve	+ve
Nerves	acute neuritis	-
hands/feet	claw hand, dropped foot	-

• **What is the main differential diagnosis of leprosy?**

- ❑ Hypopigmented macules
 - ✓ Vitiligo
 - ✓ Pityriasis alba
 - ✓ Pityriasis versicolour
 - ✓ 2ry syphilis
- ❑ Papulonodular lesions
 - ✓ Lupus erythematosus
 - ✓ Lupus vulgaris
 - ✓ Leishmaniasis
 - ✓ 2ry syphilis
- ❑ Sensory impairment
 - ✓ Causes of peripheral neuropathy

• **How would you treat leprosy?**

- ❑ Stop the infection, with antileprotic drugs
- ❑ Treat the reactions, *whenever develop*
- ❑ Educate and rehabilitate the patient
- ❑ Treat the complications
- ❑ Prevent & control the disease (early diagnosis, contact tracing, vaccination).

How can you treat a case of reactional leprosy?

- continue the chemotherapy in the usual dose
- mild reaction: add aspirin
- severe reaction: add prednisone

Can you mention some of the effective antileprotic drugs?

Drug	Action	Dose	Side effects
Dapson*	Bacteriostatic	100 mg/d	Methemoglobiemia, hemolytic anemia
Rifampicin	Bactericidal	600 mg/d	Hepatotoxicity, GIT upset, urine discoloration
Clofazimine	Anti-inflammatory	50 mg/d	Abnormal pigmentation

* contraindicated in G-6-PD deficiency

• **What is the "multi-drug therapy"?**

Using more than one drug in the treatment, *To avoid the development of resistant strains.*

• **Can you mention the WHO treatment regimen?**

Paucibacillary for <u>SIX months</u>	Multibacillary for <u>TWO years</u>
Rifampicin , once/month, <u>supervised</u>	Rifampicin , once/month, <u>supervised</u>
Dapsone , once/day, <u>unsupervised</u>	Dapsone , once/day, <u>unsupervised</u>
	Clofazimine , once/month, <u>supervised</u> once/day, <u>unsupervised</u>

- **What is the cause of cutaneous TB?**

- *M. tuberculosis*
- *M. bovis* & BCG

- **What is BCG?**

This is the abbreviation of bacillus Calmette Guerin (attenuated strain of *M. bovis*, used as vaccine).

- **Is cutaneous TB a common disease?**
NO.

- **Is the incidence increasing or decreasing?**

It was decreasing until mid 1980s.
Now it is rising up.

- **Why?**

AIDS, drug abuse, ..

- **Is there only one type of cutaneous TB?**

NO. It has a broad clinicopathological spectrum depending on:

- ✓ Infection route
- ✓ Organism virulence
- ✓ Host immunity

- **Can you mention types of cutaneous TB?**

- **Exogenous**
 - ✓ Tuberculous chancre “primary”
 - ✓ Warty
 - ✓ Lupus vulgaris
- **Endogenous**
 - ✓ Scrofuloderma
 - ✓ Orificial
 - ✓ Miliary
 - ✓ Tuberculous gumma
 - ✓ Lupus vulgaris
- **Tuberculides***
 - ✓ Micropapular
 - ✓ Papulonodular

*Immunologic reaction (in skin) to TB elsewhere in body.
TB bacilli are absent from the lesions

- **Do you know the commonest form?**

YES, Lupus vulgaris

- **In which patient it may occur?**

Those with moderate to high degree of immunity.

- **Shall you describe it clinically?**

Chronic, progressive, asymptomatic, brownish-red, soft **NODULE**, mostly on face

- **What is diascopy?**

Gentle pressure with glass slide, against the lesion will reveal **apple-jelly nodules**.

- **Can you mention the most important differential diagnosis?**

- Lupus erythematosus
- Leprosy
- Leishmaniasis
- 3ry syphilis

- **How would you treat skin TB?**

A course of anti-tuberculous drugs should be continued for **6-9 months**.

3-4 drugs are used in the 1st two months

2 drugs are enough for the remaining period

- **Would you mention some of the anti-tuberculous drugs?**

- Rifampicin
- INH
- Pyrazinamide
- Ethionamide
- Streptomycin



L. lupus, wolf to signify that the lesion looks as if bitten by a wolf

FUNGAL INFECTIONS

BASIC MYCOLOGY

- What are fungi “vegetable parasites”?

Members of “the independent kingdom of fungi”. No longer considered members of the plant kingdom. They have true nuclei (i.e. eukaryotes) and chitinous cell wall. They reproduce asexually, sexually or both. Lacking chlorophyll, they must have a source of nitrogen and carbon (i.e. being saprophytic or parasitic). They might be multicellular “mould”, unicellular “yeast” or dimorphic.

- What do you mean by a “dimorphic fungus”?

A fungus behaving as a yeast in human tissue (or culture at 37C) and as a mould in their natural habitat (e.g. soil) (or culture at 25C). They can produce deep fungal infection (e.g. *Histoplasma capsulatum*).

- What is a “dermatophyte”?

A multicellular fungus able to live on the dead animal keratin (hair, nail, skin scales)

- What is the result of that?

Superficial fungal infection {dermatophytosis, tinea or ring worm}

- How can dermatophytes do that?

- By producing an enzyme which digests keratin causing superficial skin layer to disintegrate, nail to crumble and hair to break off. (anthropophilic species)
- By producing an exotoxin or severe allergic reaction causing erythema and vesicles. (zoophilic & geophilic species)

- Is there more than “one” dermatophyte?

YES, 39 species are included in 3 genera: Microsporum, Trichophyton & Epidermophyton.

- What is the difference between?

- ❑ **Anthropophilic:** having a predilection for **humans**
- ❑ **Zoophilic:** having a predilection for **animals**
- ❑ **Geophilic:** having a predilection for **soil**

- How could you diagnose a suspected case of tinea?

- Preliminary: -Clinical picture
- Definitive : -Wood’s light
- Direct microscopy
- Culture

- How can you examine microscopically?

- Place the suspected material on a glass slide
- Add 1-2 drops of KOH 10 %
- Examine

- What do you expect to see under the microscope?

- HYPHAE**: thread-like branching filaments. Mycelia are matted hyphae.
- SPORES** : reproductive elements (RARELY seen)

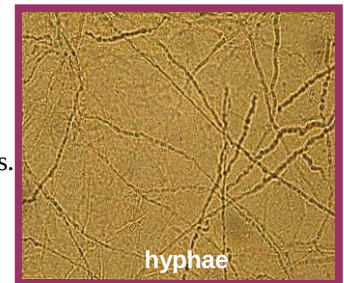
- Can you identify genera or species by direct microscopy?

NO. This can only be done by microscopy of culture products. Direct microscopy can just indicate a “fungal infection” with no further identification.

- What is the basic medium for fungal culture?

Sabouraud’s agar: a non-selective medium containing peptone, dextrose, agar & distilled water.

G. *derma*, skin
phyton, plant



What is the commonest cause of tinea?
T. rubrum

Why did they call it “ring worm”?
Because of the characteristic raised ring shape. & the ancient belief that they are caused by wormlike organisms. It has nothing to do at all with worms!!

Why did they call it “tinea”?
L. *Tinea*, clothes-moth; thought by Romans to be the cause of this disorder.

What is that?

UVR filtered through barium silicate & nickel oxide (wave length: 325-400).

It is used to diagnose and follow:

M. audouinii : bright green
M. canis : bright green
T. schoenleinii : pale green
P. ovale : yellow
C. minutissimum : coral red



TINEA

• Can you remember some of the more important DERMATOPHYTOSES?

Type / SITE	Agent	Prevalence	Differential Diagnosis	Treatment Guidelines
Tinea capitis SCALP	<i>M. canis</i> <i>M. audouinii</i> <i>T. tonsurans</i> <i>T. schoenleinii</i>	Children	-Alopecia areata -Seborrhoeic eczema -Psoriasis -Pyoderma	-Cut hair short - SYSTEMIC TOPICAL -In <u>Kerion</u> add: antibiotics, antiinflammatory
Tinea faciei FACE, smooth skin	<i>T. rubrum</i> <i>T. mentagrophytes</i>	Children	-Impetigo -Discoid L.E.	- TOPICAL
Tinea barbae FACE, beard/moustach	<i>T. mentagrophytes</i> <i>T. verrucosum</i>	Adult ♂	-Sycosis barbae	- TOPICAL
Tinea corporis BODY, except*	<i>M. canis</i> <i>T. rubrum</i> <i>E. floccosum</i>	Children	-Pityriasis rosea -Contact eczema -Impetigo	- TOPICAL
Tinea cruris GROINS	<i>T. rubrum</i> <i>T. mentagrophytes</i> <i>E. floccosum</i>	Adult ♂♣	-Candidiasis -Erythrasma -Intertrigo -Contact eczema	-Look for <i>T. pedis</i> -Avoid nylon/tight trousers -Dry properly after bath - TOPICAL
Tinea manum HANDS	<i>T. rubrum</i> <i>T. mentagrophytes</i> <i>E. floccosum</i>	Adults	-Contact eczema -Atopic eczema -Psoriasis	-Look for <i>T. pedis</i> - TOPICAL
Tinea pedis FEET	<i>T. rubrum</i> <i>T. mentagrophytes</i> <i>E. floccosum</i>	Adults	-Candidiasis -Intertrigo -Contact eczema -Psoriasis	-Dry properly after wash -Dry feet last after bath -Keep cool, wear slippers -Change socks daily - TOPICAL
Tinea unguium NAILS	<i>T. rubrum</i> <i>T. mentagrophytes</i> <i>E. floccosum</i>	Adults	-Candidiasis -Psoriasis -Lichen planus	-Look for <i>T. pedis</i> / <i>T. manum</i> -Use nail lacquer - SYSTEMIC

TOPICAL = topical antifungals (See page 23)

SYSTEMIC = systemic antifungals (See page 23)

In cases treated with **TOPICAL**, **SYSTEMIC** are added in widespread, severe or chronic cases

* any part of the body except: scalp, face, groins, hands, feet & nails.

♣ increased incidence results from: -higher incidence of tinea pedis (autoinfection)
-testicular occlusion (increases perspiration)



Tinea faciale



Tinea barbae

- What are the **hair invasion PATTERNS**?

According to the location of **arthroconidia**, relative to the hair shaft, we have **3** patterns:

Pattern	Spores	Species	Hair
Endothrix	<u>Inside</u>	<i>T. violaceum</i> <i>T. tonsurans</i>	Severe damage, breaks <u>at</u> skin surface
Ectothrix	<u>Outside</u>	<i>M. audounii</i> <i>M. canis</i> <i>T. verrucosum</i> <i>T. mentagrophytes</i>	Breaks <u>few mm. above</u> skin surface
Favic	<u>absent</u>	<i>T. schoenleinii</i>	Mild damage, considerable length

N.B. **Conidia** = asexual spores

Arthro = released by disarticulation of hyphae

In all the **three** patterns, **hyphae** are present within the hair shaft

In **ectothrix**, the **arthroconidia** may be: -small “microconidia”
or -large “macroconidia”

In **favus**, characteristic **air spaces** are present within hair shafts

G. thrix, hair

- Can you mention the **clinical** patterns of tinea capitis?

Pattern	Species	Presentation
☐ Non-inflammatory		
Scaly *	<i>M. canis</i> <i>M. audounii</i>	One or more, round or oval, scaly “ grey patch ” of partial alopecia, with lusterless broken-off hair
Black dots	<i>T. violaceum</i> <i>T. tonsurans</i>	Broken-off hair at the scalp surface producing “ black dots ” within alopecic areas
☐ Inflammatory		
Kerion ♥	<i>T. verrucosum</i> 2ry: staph. aureus	Red, tender, boggy swelling, studded with broken loose hair. Purulent ooze from follicles. Occipital lymphadenopathy
Favus ♣	<i>T. schoenleinii</i>	Scutula: cup-shaped, honey-coloured, mucous-smelling crusts of matted mycelia and epithelial debris

*The commonest, involutes spontaneously at puberty, non-scarring alopecia

♥Clinical manifestation of host immune response, scarring alopecia

♣The rarest, does not involute at puberty, scarring alopecia

- Would you describe the clinical picture of tinea corporis?

A round, oval or semicircular scaly **PATCH** with a sharp, slightly raised, slightly vesicular border. Slow peripheral spread and central clearing will result in a typical ring-like pattern.

- Can you mention the primary lesion of tinea cruris?

Bilateral, fan-shaped, red, scaly **PATCH** with a sharp, slightly raised, slightly vesicular border.

- What about the secondary changes?

Oozing, crusting, oedema. Lichenification occurs in chronic cases.

فمالي وللدنيا طلابي نجومها *** ومسعاي منها في شقوق الأرقام

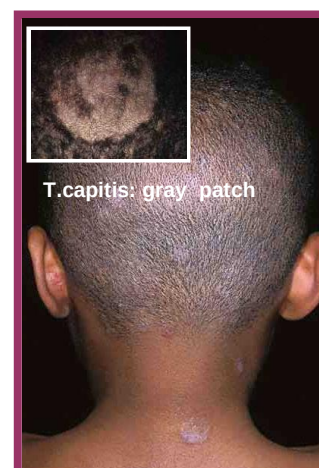
من الحلم أن تستعمل الجهل دونه *** إذا اتسعت في الحلم طرق المظالم

وأن ترد الماء الذي شطره دم *** فتسقى إذا لم يسق من لم يزاحم

وبالناس روى رحمه غير راحم *** ومن عرف الأيام معرفتي بها

فليس بمرحوم إذا ظفروا به *** ولا في الردى الجاري عليهم بآثم

المتني



kerion

• What are the clinical types of tinea pedis?

- ❑ **Interdigital:** maceration, fissuring, scaling of web spaces *the most common “athlete’s foot”*
- ❑ **Vesiculobullous:** pruritic tense vesicles and bullae of the sole
- ❑ **Moccasin:** erythema, scaling of the sole

Does it affect only athletes?
NO. It can affect even the laziest people!!

• What are the clinical types of tinea manum?

- ❑ Dry, scaly **patch**, almost always unilateral *the most common*
- ❑ **Vesiculobullous**

• Is there a difference between tinea unguium and onychomycosis?

- ❑ **Tinea unguium:** infection of the nail plate by dermatophytes
- ❑ **Onychomycosis:** infection of the nail plate by moulds (including dermatophytes) or Candida.

• What are the clinical types of onychomycosis?

- ❑ Distal subungual *T. rubrum*
 - ❑ Proximal subungual *T. rubrum*
 - ❑ Superficial white *T. mentagrophytes, moulds*
 - ❑ Candidal *C. albicans*
- more common in the **toe** nails.
- more common in the **finger** nails



CANDIDIASIS

• What is the most common causative agent of candidiasis?

C. albicans. Normally, it is a part of GIT microflora.

• Shall you mention some clinical presentations of superficial candidiasis?

Syndrome	Site	
Thrush	Oral mucosa	White, adherent, cheese-like plaques
Perleche	Mouth angles	Erythema, fissuring, creamy exudate
Interdigital	Web spaces	Erythema, fissuring, maceration
Paronychia	Proximal nail folds	Erythema, induration, purulent discharge?
Intertrigo	Skin folds	Erythema, superficial pustules, thin creamy exudate

• Do you know some of the predisposing factors for candidiasis?

- ❑ **Local:** occlusion, moisture, warm temperature (**factors damaging stratum corneum**)
- ❑ **Systemic:** immunosuppression, endocrinopathy, drugs (antibiotics, oral contraceptives), pregnancy

ANTIFUNGAL AGENTS

- Shall you mention some of the **TOPICAL** antifungal agents?

GROUP/Agent	Action	Spectrum	Indication
AZOLES ♣ Miconazole Clotrimazole Ketoconazole	Fungicidal	C, D, M	- Dermatophytoses, Candidiasis, Pityriasis versicolor
ALLYLAMINES ♣ Terbinafine Naftifine	Fungicidal	D	-Dermatophytoses
POLYENES ♣ Nystatin	Fungicidal	C	-Candidiasis
OTHERS Ciclopirox♣ Amorolfine♣ Selenium sulphide Whitfield's ointment♥ Castellani's paint¶	Fungicidal Fungicidal Unknown Fungistatic Antimicrob.	C, D, M C, D, M M D D, C	-Onychomycosis -Onychomycosis -Pityriasis versicolor -Tinea pedis -Intertrigo, Acute candidal paronychia

N.B. C= Candidia, D= Dermatophytes, M= Malassezia

♣**Act on:** cell membrane

***Azoles** may be: fungicidal (when applied topically "high concentration", or: fungistatic (when given systemically "low concentration")

♥Also: keratolytic

¶Also: drying, local anaesthetic



Onychomycosis



Candidiasis

- What are the indications of systemic antifungals?

- ❑ Systemic fungal infections
- ❑ Superficial fungal infections, under certain conditions:
 - ✓ **scalp, nails** lesions
 - ✓ **widespread, severe or chronic** lesions

- Can you mention some of the more important **SYSTEMIC** antifungals♣?

GROUP/Agent	Dose	Action	Spectrum
GRISEOFULVIN *	10 mg/kg/d	Fungistatic	Dermatophytes
ALLYLAMINES Terbinafine	250 mg/d	Fungicidal	Dermatophytes
AZOLES Fluconazole Itraconazole Ketoconazole♥	150 mg/d 100 mg/d	Fungistatic	Candida Broad spectrum

♣All of them are -Associated with GIT upset, headache, rash
-Contraindicated in hepato-renal failure and pregnancy

* **Incorporated** in newly formed host keratin

Acts on dermatophyte nucleus (antimitotic)

Dosage should be taken after fatty meals

Contraindicated: in porphyria & SLE.

Interacts: with warfarin

♥It has been largely supplanted by itraconazole { due to ↓ selectivity, penetration & ↑ side effects (antiandrogen, hepatotoxic) }

Pityriasis versicolor

What is its correct name?
Pityriasis versicolor.

- Is “tinea versicolor” a “TRUE” tinea?

NO. It is a superficial fungal infection caused by the lipophilic yeast *Malassezia furfur*. On the contrary, “true” tineas are caused by keratinophilic moulds (i.e. dermatophytes)

- Is it an infectious disease?

NO. *Malassezia furfur* { synonym: *Pityrosporum (orbiculare or ovale)* } is one of the resident skin flora. Under certain conditions, it changes to the pathological filamentous (i.e. mycelial) form.

Mention some conditions predisposing to such pathological change

- ❑ High temperature
- ❑ Hereditary factors

Do you know other diseases that might be caused by this fungus?

- ❑ Pityrosporum folliculitis
- ❑ Seborrhoeic dermatitis

- What is the clinical picture of pityriasis versicolor?

Sharply demarcated MACULES and patches, covered with fine branny scales. The lesions may be hypo / hyperpigmented (this is why it is “**versi**”!!), mostly on the upper trunk. **Wood’s light**: yellow fluorescence

- Would you mention the differential diagnosis?

- ❑ Vitiligo
- ❑ Pityriasis alba
- ❑ Pityriasis rosea
- ❑ Tinea corporis

Why hypopigmented?
-disturbed melanogenesis

Why hyperpigmented?
-hyperkeratosis
-matted fungus

- Which lab. tests can be used?

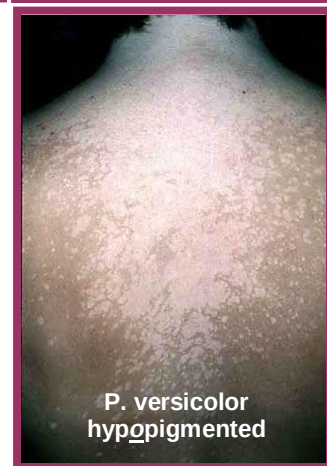
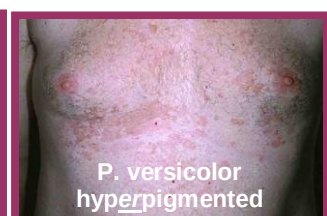
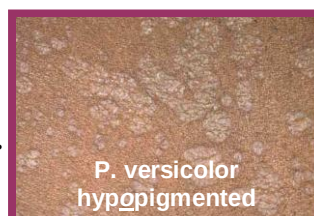
Direct Microscopy : short hyphae and spherical budding cells “spaghetti and meat balls”.

- Can thorough washing treat it?

NO.

- Mention some of the treatment options.

- ❑ **Topical :**
 - ✓ Imidazoles creams, lotions or shampoos
 - ✓ Selenium sulphide shampoo
 - ✓ Zinc pyrithione shampoo
- ❑ **Systemic:**
 - ✓ Imidazoles e.g. ketoconazole
 - ✓ Triazoles e.g. itraconazole



- Is it common to relapse after treatment?

YES, very common.

- Does treatment rapidly correct depigmentation?

NO, this takes several months.

- Does tanning help repigmentation?

NOT at all. It makes the condition worse (only normal skin around lesions will pigment)

INFESTATIONS

SCABIES

L. *scabo*, to scratch

- **What is scabies?**

A fairly common, highly contagious, parasitic skin disease.

- **Do you know its cause?**

YES, the mite *sarcoptes scabiei*. (acarus)

- **Who is especially at risk?**

Poorer, overcrowded populations, with lack of hygiene e.g. prisoners, soldiers at war.

- **Is it exclusive for humans?**

No. It can infest about **40** different species. Each host species has its own variant e.g. var. *hominis* for humans.

- **Where does the mite live?**

The entire life cycle of var *hominis* is on the human skin.

After copulation, ♀ mite burrows in the stratum corneum. Eggs laid down (40-50/life cycle) hatch after 3-4 days. The adult ♀ mite dies after one month, while the ♂ dies immediately after copulation.

- **How long is the incubation period** (for the **primary** infection)?

It is **4-6** weeks (so that sensitisation can occur)

- **How many mites are present on the skin?**

About **10-15** adult ♀.

- **How is it transmitted?**

Direct, prolonged, close contact with infested persons is the most important mode.

Is it an **STD**?
YES ...!!

Why is it so difficult to be transmitted by indirect methods?

The mite can not live away from the host more than few days (2-5 days).

- **How can you diagnose it?**

□History:-Nocturnal pruritus

-Other family members affected

□Exam :-Characteristic lesions at characteristic sites (page 26)

□Lab. :-Burrow scrapping **to reveal**: ♀ mite, eggs or faeces under the **microscope**.

- **What is the cause of pruritus?**

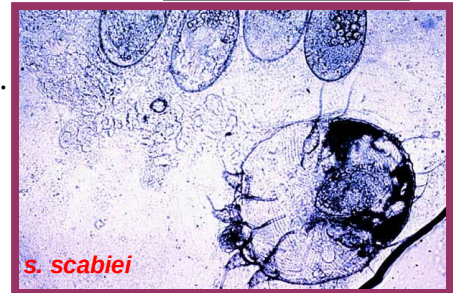
It is due to hypersensitivity to the mites or their products e.g. eggs, faeces

- **Why is it nocturnal?**

Still uncertain. Most probably it is due to host factors (e.g. lower itch threshold upon relaxation).

- **Is it related to diurnal activity of the mite?**

NO.



- Is it necessary to ask every patient if he has contacted an animal e.g. dog, cat, etc....?
YES.

- Why?

Although animal variants of *sarcoptes scabiei* **CANNOT** infest humans (no copulation or burrow formation), they **CAN** live for a short period on human skin leading to pruritic papulo-vesicles, at the site of animal contact (hypersensitivity reaction). It is self-limited.

- What are the characteristic lesions of classical scabies?

Primary : -**BURROW**

Secondary: -papules, scratch marks, excoriations

Burrow: linear epidermal tunnel caused by a burrowing parasite, small vesicle overlies the site of the ♀ mite

- What are the preferred sites?

- Interdigital webs of hands, Flexor wrist, Extensor elbow
- Axilla, Nipple, Central abdomen
- Genitalia, Buttocks, Innear thigh

- Can you enumerate some of the clinical variants?

- Classical** (mentioned above)
- Recurrent** {short IP (1-4 days), few lesions}
- Infantile** (atypical distribution: *palms, soles, face, scalp*, Vesiculobullous eruption)
- Animal** (mentioned above)
- Norwegian**
- Nodular** (indurated brownish-red nodules, axilla-genitalia-groins, severe hypersensitivity, no mites)



- How many mites could be present in Norwegian scabies?

Up to 2 millions !!!

Do you have an explanation?

Norwegian scabies is mainly prevalent among immunosuppressed and mentally retarded.

- Shall we expect horrible pruritus?

Paradoxically, itching is either absent !! or minimal.

- What is the danger of Norwegian scabies?

It can initiate epidemics (innumerable mites, in non-complaining persons)

- Would you mention some of scabies' complications?

- Insomnia
- Secondary infection (glomerulonephritis may follow)
- Contact eczema
- Acarophobia

- What is the main differential diagnosis of scabies?

- ☐ Papular urticaria
- ☐ Lichen planus (L.P.)
- ☐ Eczema
- ☐ Dermatitis herpetiformis (D.H.)
- ☐ Pediculosis corporis
- ☐ Parasitophobia



- Is it a self-limited disease?

NO. It persists indefinitely till treated. However number of mites decreases in chronic cases.

• **What are the lines of treatment?**

- **Curative** : Antiscabitics
- **Supportive, Symptomatic** : Antipruritics & antibiotics
- **Preventive** : Contact tracing and treatment

• **Can you mention some of the antiscabitics?**

Agent	How to use	Comments
Permethrin cream, 5%	10 hours x 1*	drug of choice , relatively expensive, contraindicated in neonates & pregnancy
Hexachlorocyclohexane♣ lotion, 1%	8 hours x 1*	neurotoxic, contraindicated in infants, children & pregnancy
Sulphur precipitate, 5-15%	24 hours x 3	drug of choice in neonates & pregnant messy, offensive
Benzyl benzoate emulsion, 25%	24 hours x 2	very irritant
Malathion lotion, 0.5%	12 hours x 1	highly toxic
Crotamiton Cream/lotion, 10%	24 hours x 3	also antipruritic, low efficacy (30% resistance)

* Most patients respond to one treatment. Repeat after one week if there is microscopic or morphologic evidence of ttt failure.

♣ Previously known as **gamma benzene hexachloride**, but now it is a misnomer that should be dropped

• **What should be done BEFORE applying the antiscabitic?**

- Cut the fingernails & toenails short
- Remove rings and watchbands
- Take a warm shower, then dry with a clean towel

• **Where is the treatment applied?**

- **Infants**: **all over**, including the face and scalp
- **Others**: **starting from** the area behind the ear **down**

• **What should be done AFTER applying the antiscabitic?**

- Leave the medication, for the number of hours suggested
- Rinse it off thoroughly, with tepid (i.e. slightly warm) soapy water
- Dry the skin
- Disinfect clothes and furniture:
 - ✓ underwear, sleepwear, sheets, pillowcases and towels should be boiled at 50°C for 10min
 - ✓ outerwear and other furniture can be left unused for few weeks, (mite dies away from host)

• **How could you explain persistent itching?**

- Failed treatment
- Hypersensitivity to treatment
- Cross reaction between *S. scabiei* and house dust mite
- Acarophobia
- Reinfestation

What is its commonest cause?

Failure to cut nails short & apply treatment beneath

What is the commonest cause of reinfestation?

Undetected untreated contacts.

PEDICULOSIS

L. pediculus, louse (pl. lice)

- **What are pediculi?**

Blood-sucking insects, infesting the skin, causing considerable irritation.

Two species of this ectoparasite can affect humans:

- ✓ *Pediculus humanus* var. *capitis* common among school children
- var. *corporis* common among vagrants (**otherwise so rare**)
- ✓ *Phthirus pubis* usually **STD**

- **What is the clinical presentation of pediculosis CAPITIS?**

□History: - Persistent pruritus, at **SCALP**: back & sides

□Exam. : - **Nits** “eggs”, attached to the hair shaft

- **Lice**, can be seen by the naked eye when they move
- Crusting, lymphadenopathy (with secondary bacterial infection following scratching)

What is vagabond's disease?

Depigmented macules within diffuse hyperpigmented patches over waist, axillae, groin, back, neck. It affects older, ill people with poor hygiene or alcoholics. It is usually associated with ectoparasitosis

- **What is the clinical presentation of pediculosis CORPORIS?**

□History: - Persistent intense pruritus in skin areas closest to clothes (e.g. shoulders, breasts, buttocks)

□Exam. : - Urticated papules, extensive scratch marks & excoriations; infection, lichenification

- **Lice**, can be seen in clothes (specially seams of underwear)

why?

Although it feeds off the patient, it lives and breeds in clothes and bed linings !!

- **What is its differential diagnosis?**

- Scabies
- Flea bites
- Bed bug infestation

- **What is the clinical presentation of pediculosis PUBIS?**

□History: - Persistent intense pruritus in affected sites

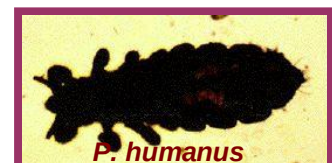
□Exam : - **Lice & nits** can be seen mainly in the pubic hair, may spread up to eyelashes and eyebrows, but **NEVER** in the scalp.

- **What are the treatment measures?**

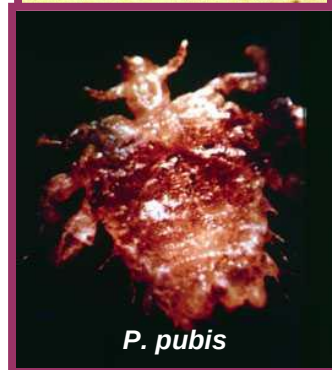
- Cut hair short, *if possible*
- Apply **pediculocidal** agents
- Disinfect clothes, or use new ones (specially with corporis)
- Remove nonviable nits (capitis) {with a fine-toothed comb, after rinsing the hair with 8% formic acid solution} to destroy the cement-like chitin adhering them to hair.

- **Shall you mention some pediculocidal agents?**

- Hexachlorocyclohexane**, lotion 1%, applied for 24 hours, repeated after 10 days.
- Permethrin**, lotion 5%, applied for 24 hours.
- Malathion**, lotion 0.5%, applied for 24 hours, in resistant cases.



P. humanus



P. pubis

LEISHMANIASIS

- **What is leishmaniasis?**
A wide spectrum of chronic, granulomatous, protozoal diseases.
The clinicopathological condition depends on the causative agent and the host immunity.
- **What is its causative agent?**
Several species of the genus *Leishmania* e.g. *L.tropica*, *L.major*.
- **How is it transmitted?**
Biting sand flies (*Phlebotomus* species) spread the disease between humans and a large variety of animal reservoirs e.g. rodents, dogs
- **What is the clinical spectrum?**
 - ❑ Cutaneous: localised or diffuse (nodules, ulcers, crusts, scars)
 - ❑ Mucocutaneous
 - ❑ Visceral
- **Where is it prevalent?**
Widespread. Cutaneous types are most common in the Middle east and Mediterranean area.
- **Is it easy to treat it?**
NO, in spite of the wide variety of treatment options e.g. **antimonials, rifampicin, cryotherapy**.

خير الكلام

عَنْ رَسُولِ اللَّهِ - صَلَّى اللَّهُ عَلَيْهِ وَسَلَّمَ - أَنَّهُ قَالَ:

مَا مِنْ أَمْرٍ يَخْذُلُ أَمْرًا مُسْلِمًا فِي مَوْضِعٍ تُنْتَهَكُ فِيهِ حُرْمَتُهُ وَيُنْتَقَصُ فِيهِ مِنْ عَرْضِهِ إِلَّا خَذَلَهُ اللَّهُ فِي مَوْطِنٍ يُحِبُّ فِيهِ نَصْرَتَهُ وَمَا مِنْ أَمْرٍ يَنْصُرُ مُسْلِمًا فِي مَوْضِعٍ يُنْتَقَصُ فِيهِ مِنْ عَرْضِهِ وَيُنْتَهَكُ فِيهِ مِنْ حُرْمَتِهِ إِلَّا نَصَرَهُ اللَّهُ فِي مَوْطِنٍ يُحِبُّ نَصْرَتَهُ (ابن داود)

الْقَضَاءُ ثَلَاثَةٌ ، اثنان في النَّارِ وَوَاحِدٌ في الْجَنَّةِ : رَجُلٌ عَلِمَ الْحَقَّ فَقَضَى بِهِ فَهُوَ فِي الْجَنَّةِ وَرَجُلٌ قَضَى لِلنَّاسِ عَلَى جَهْلٍ فَهُوَ فِي النَّارِ وَرَجُلٌ جَارَ فِي الْحُكْمِ فَهُوَ فِي النَّارِ (ابن ماجه)

مَنْ أَعَانَ عَلَى خُصُومَةٍ بَظْلِمٍ - أَوْ يُعِينُ عَلَى ظُلْمٍ - لَمْ يَزَلْ فِي سَخَطِ اللَّهِ حَتَّى يَنْزِعَ (ابن ماجه)

What is papular urticaria?

An allergic reaction to insect bites.
Recurrent crops of itchy, grouped,
Small, excoriated, urticated, papules.
Mostly on the arms and legs.

Treatment involves:

- ✓ insect control
- ✓ systemic antihistamines
- ✓ topical corticosteroids

What is prurigo?

Intensely pruritic papules, without
an identifiable local cause.

Who is Hebra?

Ferdinand von Hebra, an Austrian dermatologist, 1816-1880. He described many skin diseases including a type of prurigo, named after him. It was common in Vienna in the last century. Most of the patients had pruritic papules over the extremities' extensors, usually starting in the childhood. Some were atopic. Others were suffering poor hygiene. Insect bites or parasitosis had been claimed. Currently it is a questionable term which is now rarely diagnosed. It had not been reviewed after Koscard in 1962.

ACNE

G. *akmē*, the highest point
It was copied incorrectly as acne

- **What is acne?**

Chronic inflammatory disorder of pilosebaceous apparatus, leading to greasiness and polymorphic eruption.

- **How common is it ?**

Perhaps it is **the most common** skin disorder.
Almost, every one has got some kind of acne, sometime during his life.
Both sexes are affected. It starts earlier in ♀ and peaks at **16-19** years.

- **Shall you mention some acne types?**

- Vulgaris
- Drug-induced
- Conglobata: abscesses/cysts, interconnecting sinuses leading to severe scarring
- Fulminans: Acne conglobata + constitutional manifestations
- Excoriée: due to obsessional picking in young girls

- **What is its pathogenesis?**

MULTIfactorial:

- Increased production of **abnormal** sebum
- **Abnormal** keratinization of follicular duct, with subsequent obstruction
- Presence of *Propionibacterium acne*; an anaerobic diphtheroid
- Increased sensitivity of androgen **receptors**, in sebaceous glands

Which organisms are present in acne pustules?

NON. Usually, it only contains sterile sebum and neutrophils.

The interplay of the above mentioned factors will lead to:

follicular plugging, activation of *P. acne* and release of inflammatory mediators.

- **What are the common MYTHS regarding the aetiology of acne?**

- Chocolates and fatty acids cause acne
- Poor hygiene produces acne
- Masturbation produces acne
- Sunlight exposure improves acne

- **Do any drugs cause or aggravate acne?**

YES, e.g. Steroids, Halogens, Antidepressants, Antiepileptics

- **What is the clinical presentation of the common type “acne vulgaris”?**

- **Complaint:** - Emotional upset (common)
- Tenderness of large pustules, itching (rare)
- **Exam** : - non-inflammatory lesions: **comedones** (*primary lesion*)
- inflammatory lesions : -superficial **papules, pustules**
-deep **nodules, cysts**
-sequela : **scars** (atrophic/hypertrophic)

Comedo: collection of sebum and keratin, plugged in a pilosebaceous follicle

- closed comedos i.e. **white** heads (deep plug)
- open comedos i.e. **black** heads (superficial plug)

L. comedo, to eat up
(Pl. comedones)

- **What are the most commonly affected sites?**
FACE, shoulders, upper chest, back.

• What is the differential diagnosis of acne?

- Other causes of folliculitis
- Rosacea

What is rosacea?

Skin disease of unknown aetiology, **mostly affecting** facial convexities of adult ♀.

It **presents** with - *fixed* erythema & telangiectasia
- *fluctuating* follicular papules & pustules.

Severe late cases may develop rhinophyma
(hyperplasia of sebaceous glands and connective tissue on the nose).

Treatment : - Topical: metronidazole, sulphacetamide + sulphur
- Systemic: tetracycline, isotretinoin.



• Why should we treat acne?

- To prevent scarring.
- To avoid psychological disturbances of the patient.

What is the psychological impact of acne?

- Inferiority
- Anger
- Depression
- Embarrassment
- Poor academic performance
- Unemployment

• How shall you treat it?

- **TOPICAL** measures (more effective on the face)

alone, **for** mild adolescent cases (comedones, papules)

adjuvant to systemic measures, **for** other cases

- Regular gentle cleansing with soap and water
- **Benzoyl peroxide** (bactericidal, comedolytic)
- **Retinoids** e.g. adapalene, tretinoin (normalises follicular keratinisation)
- **Azelaic acid** (antiinflammatory, anticomedonal)
- **Topical antibiotics** e.g. clindamycin, erythromycin (antiinflammatory)
- **Sulphur**
- **Cosmetic camouflage**

How can we treat **acne scars**?

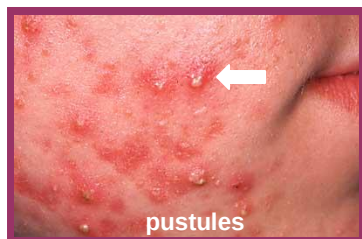
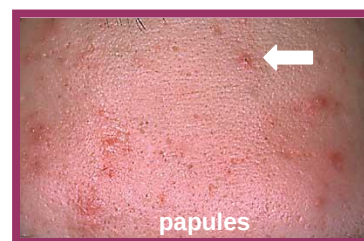
- **Laser**; resurfacing
- **Collagen**; injection

- **SYSTEMIC** measures

for - Moderate to severe cases (painful papules, nodules, cysts)

- Patients with low morale

- **Antibiotics** e.g. tetracycline, erythromycin
- **Retinoids** e.g. isotretinoin
- **Hormones** e.g. antiandrogen/estrogen (in ♀)



Acne vulgaris

ECZEMA

G. *eczema*, to boil over

- **What is eczema?**

An inflammatory reaction pattern of the skin. It is a kind of dermatitis. However, not all dermatitis are eczemas.

- **What do you mean by “reaction pattern”?**

Various skin compartments interconnect anatomically & interact functionally, behaving as “reactive units” in response to different stimuli producing the so-called reaction pattern e.g. eczema, urticaria. Reaction pattern is different from Disease (which has certain common etiology, pathology, clinical picture and effective treatment) as well as from Syndrome (a constellation of symptoms and signs).

- **What are the main features of eczemas?**

- Acute: erythematous, ill-defined patch of papules, vesicles & bullae with oozing and crusting
- Chronic: scaling, fissuring & lichenification (Page

What is its histopathological hallmark?
Spongiosis: intercellular oedema.

- **How can you treat it?**

General principles

- **Explanation, reassurance**
- **Symptomatic measures:**
 - ✓ Antipruritics
 - ✓ Antibiotics, *if secondarily infected*
- **Specific measures:**
 - ✓ **Acute:**
 - Wet dressing e.g. k permanganate solution
 - Steroid cream or lotion
 - ✓ **Subacute:**
 - Steroid cream or lotion
 - ✓ **Chronic:**
 - Steroid ointment
 - Hydration
 - Cut nail short

How are eczemas classified?

According to aetiology:

- **Exogenous** (contact)
 - irritant
 - allergic
 - photodermatitis
- **Endogenous** (constitutional)
 - atopic
 - seborrhoeic
 - discoid
 - pompholyx
 - stasis

According to clinical picture:

- Acute
- Subacute
- Chronic

- **What is irritant contact dermatitis?**

Eczema **produced by** exposure to an irritant*.

It is **common** in certain job e.g. building, cleaning, hair dressing.

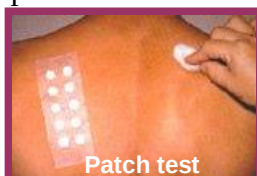
Treatment depends mainly on avoiding exposure to the responsible substance

- **What is allergic contact dermatitis?**

Eczema **due to** delayed (type IV) hypersensitivity.

Following contact with the skin, a hapten forms a protein complex that is presented to lymphocytes by Langerhans cells. Further exposure, will lead to elicitation of eczematous reactions, specific to one chemical or its close relatives.

It may involve any skin site. **Desensitization** is rarely possible.



Patch test



Allergic CD
(brasiers)

* A substance that causes an inflammatory response in almost all individuals when applied in sufficient concentration for enough time e.g. detergents, alkalies, weak acids

- **What is ATOPY?**

A genetically-determined increase in the level of IgE in response to suitable triggers e.g. irritants, dryness, emotional stress. Atopic subjects may or may not develop one or more of clinical diseases e.g. atopic dermatitis, asthma, allergic rhinitis.

How common is it?
15% of the population have at least one atopic manifestation. The prevalence is increasing.

L. *atopos*, without a place to signify lack of apparent link between affected sites

- **What are the cardinal features of atopic dermatitis?**

- ❑ Intense **itching/scratching**
- ❑ Typical **morphology/distribution**
- ❑ Chronic/relapsing **course**
- ❑ Personal/family **history**

How does it present?

The clinical lesions differ according to the patient's age:

- Infants:** vesicles, oozing, crusting {face (diapers are spared)}
- Children:** leathery, dry, excoriated {cubital & popliteal}
- Adults:** more lichenification {cubital & popliteal}

- **What is the prognosis?**

2/3 of cases remit before the age of 10 years.

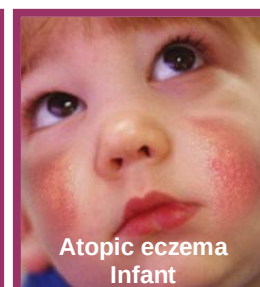
- **How shall you treat it?**

(in addition to the general principles mentioned before)

- ❑ **Avoid** triggers
- ❑ **Use** emollients
- ❑ **Try** PUVA, cyclosporine, Chinese herbs



Atopic eczema
Child



Atopic eczema
Infant

- **What is SEBORRHOEIC dermatitis?**

Subacute eczema characterised by: well-defined erythematous patches with yellowish greasy scales, characteristically distributed over the seborrhoeic sites (sites of maximal sebaceous gland concentration) .

L. *sebum*, suet, tallow شحم
G. *rhoia*, a flow

Can you mention some such sites?

Scalp, eyebrows, nasolabial folds, upper trunk

- **Do you know its exact cause?**

No, it is unknown. However, several *theories* are suggested:

- ❑ Altered sebum amount or composition
- ❑ *Pityrosporum*
- ❑ CNS disorders

Is it contagious?
No !!!

- **How does it present?**

- ❑ **Infants:** Cradle cap
- ❑ **Adults:** Scalp (pityriasis capitis): - pityriasis sicca (dandruff)
- pityriasis oleosa (oily)

Does it lead to permanent hair loss?
No.

Trunk: - petaloid (small yellowish red follicular papules, covered with greasy scales. Confluence lead to lesions which resemble flower petals)

- pityriasiform (oval, papulosquamous eruption, resembling pityriasis rosea)

G. *petalon*, a thin leaf

- **Can you tell me the main treatment options?**

- ❑ Scalp lesions: tar, selenium sulphide, zinc pyrithione, ketoconazole **SHAMPOO**
- ❑ Trunk lesions: hydrocortisone, pramoxine, ketoconazole **CREAM**
- ❑ Resistant: UVB, ketoconazole **TABLETS**
- ❑ Generalised: isotretinoin **TABLETS**

- **What is?**

Discoid eczema: chronic, multiple, coin-shaped, highly itchy plaques, <5cm, on limbs of mid-aged ♂.

Pompholyx: recurrent attacks of deep vesicles on the palms, fingers, soles of adults; lasting few weeks

Stasis eczema: chronic patchy eczema of lower legs, due to chronic venous insufficiency, in people whose work require long hours of standing e.g. barbers.

You may find: oedema, varicosity, haemosiderin deposits, ulcers.

URTICARIA

- **What is urticaria?**

A vascular reaction pattern. Its hallmark is **wheals**: flat-topped papules/plaques, with sharp, unstable borders. Characteristically **evanescent** i.e. disappear within hours.

- **How does it occur?**

Release of certain mediators e.g. *histamine*,
Capillary dilatation & increased permeability
Extravasation of protein-rich fluid
Upper dermal oedema

The histopathological hallmark

The definite factors, which provoke the release of mediators, are largely **unknown** (75%).

However, in the **known** cases, they might be either:

- **Immunologic** (5%)
- **Non-immunologic** (20%)

- **How common is urticaria?**

15-20% of the population will experience at least **one** episode of urticaria, during their life time.

- **What is the chronic urticaria?**

Widespread wheals, lasting for <24 hours, recurring almost daily, for, at least, six months.

- **What is the most common type of physical urticaria?**

- **Cholinergic**: due to high acetyl choline secretion after anxiety, exercise
- **Dermographism**: exaggerated triple response after rubbing, scratching

- **What is angioedema?**

Deep urticarial reaction, involving mainly the subcutaneous tissue.

Mostly at skin-mucosal junctions {with loose dermis & subcutaneous tissue (e.g. peri-oral, peri-orbital, genital)}

What are the causes of urticaria?

-Immunologic (5%)

-IgE-mediated (type I hypersensitivity)

-specific antigen sensitization

the allergen may be:

- Infectant e.g. sinusitis
- Infestant e.g. ascariasis
- Injectant e.g. serum
- Ingestant e.g. seafoods
- Inhalant e.g. pollens

-atopic diathesis

-contact urticaria

-Physical urticaria

-Cold

-Heat

-Pressure

-Vibration

-Light

-Water

-Exercise

-Complement-mediated

-angioedema (hereditary/acquired)

-serum sickness

-Non-immunologic (20%)

-Mast cell secretagogues:

- curare
- radiocontrast media
- opiates

-Agents altering arachidonic acid

- Chemicals e.g. benzoate
- Drugs e.g. aspirin



How can you treat urticaria?

-**Preventive measures**: avoid aspirin in any form

-**Curative measures**: find and avoid causes

-**Symptomatic measures**:

- Antihistamines (the mainstay)
- Sympathomimetics (β -agonists)
- Systemic steroids (rarely needed)
- Attenuated androgens

DRUG REACTIONS

- **What is a drug?**

A chemical substance made available for an intended purpose, for the benefit of the patient.

- **What is the safest method of drug administration?**

Oral.

- **What is the most risky method?**

Intra Muscular.

- **What is the drug reaction?**

Undesirable, usually unexpected, response to the drug, independent on the intended purpose of the medication.

- **What are its types?**

Non-allergic	Allergic
❑ Often predictable ❑ The majority (90%) ❑ Affecting almost all patients taking the drug at <u>sufficient dose</u> for a sufficient time ❑ e.g. overdose, cumulation, interaction, idiosyncrasy, teratogenicity	❑ Less predictable ❑ The minority (10%) ❑ Dose-<u>in</u>dependent ❑ e.g. ✓ IgE-mediated (type I) ✓ Cytotoxic (type II) ✓ Immune complex-mediated (type III) ✓ Cell-mediated (type IV)

- **What is the most common form of drug reactions?**

Drug **eruptions**.

- **What is the drug eruption?**

A drug reaction primarily involving the skin.

What is its most common form?
The morbilliform (maculopapular)

Would you give me SOME examples?

-Acneiform e.g. **corticosteroids**
 -Psoriasiform e.g. **lithium**
 -Lichenoid e.g. **gold**
 -Bullous e.g. **barbiturates**
 -Eczematous e.g. **methyldopa**
 -Pigmented e.g. **contraceptive pills**
 -Ichthyotic e.g. **carbamazepine**
 -Fixed drug eruption

What are the commonest offenders?

-Antibiotics e.g. penicillins, sulphonamides
 -NSAIDs e.g. salicylates
 -CVS drugs e.g. β -blockers

What is the absolute commonest offender?
Amoxycillin is the cause in >50% of cases

What is that?

Round, erythematous/purple, plaque (may be bullous)
 It recurs at the same site, each time drug is taken, pigmentation persists between acute episodes.
Sulfonamides are major cause.

ERYTHEMA, PURPURA

- What is **erythema**?

Redness of the skin

G. *erythēma*, flush

- What produces it?

Capillary dilatation and congestion, mostly secondary to inflammation e.g.

- ❑ Drugs
- ❑ Infections
- ❑ Collagen diseases

What is the most common cause of erythema?
IDIOPATHIC !!!

- Can you mention its types?

- ❑ Diffuse e.g. *scarlatiniform*: punctate with dark red follicular ostia, (sand paper skin)
morbiliform: net-like with irregular macules, slightly elevated papules
roseolar
- ❑ Localised e.g. palmar
- ❑ Figurate: chronic erythematous eruptions made up of bizarre serpiginous rings.
Unfortunately they mostly have latin labels e.g.
-erythema annulare centrifugum
-erythema chronicum migrans
-erythema gyratum repens
-erythema marginatum rheumaticum

What is Erythema Multiform?

Acute, self-limited, cytotoxic reaction pattern of the skin, **induced by** several factors as: infections e.g. *herpes simplex* & drugs e.g. *sulphonamides*.

It **consists of** polymorphic rash (macules, papules, vesicles). The most characteristic is the target (iris / bull's eye) lesion. This consists of 3 zones (dark or blistered center, surrounded by a 2nd pale zone. The 3rd one is a rim of erythema)

Distribution is symmetrical on the extremities.

Stevens Johnson's syndrome:

An extreme febrile form of EM, with erosion & haemorrhagic crusts involving lips & other mucous membranes. It might be fatal.

- What is **purpura**?

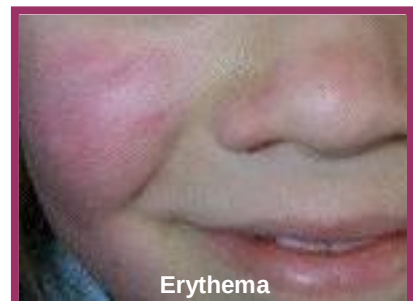
G. *Porphyra*, purple

Skin discolouration due to RBCs extravasation

- What are the causes of purpura?

- ❑ **INTRAVASCULAR**
 - ◆ **Abnormal platelet number**
 - ↓ **number (thrombocytopenia)**
 - decreased production
 - drugs
 - radiation
 - increased destruction
 - idiopathic thrombocytopenic purpura
 - disseminated intravascular coagulopathy
 - sequestration
 - splenomegaly
 - ↑ **number (thrombocytomia)**
 - malignancy
 - ◆ **Abnormal platelet function**
 - congenital** Von Willebrand disease
 - acquired** Systemic lupus erythematosus
- ❑ **VASCULAR**
 - ◆ Vasculitis
 - ◆ Cryoglobulinaemia
 - ◆ Emboli
- ❑ **EXTRAVASCULAR**
 - ◆ Steroids
 - ◆ Senility

Petechiae: small pinpoint purpuric spots.
Ecchymoses: larger bruise-like purpuric lesions.



Erythema



Purpura

PAPULOSQUAMOUS DISEASES

- **What is the meaning of “papulosquamous”?**

Scaly erythematous inflammatory papules and plaques.

- **Can you mention some examples?**

- ❑ Psoriasis
- ❑ Pityriasis rubra pilaris (PRP)
- ❑ Lichen planus
- ❑ Pityriasis rosea
- ❑ Secondary syphilis

Is it common?

YES. It affects 2% of the population worldwide.
Highest incidence is in Scandinavia.

- **What is psoriasis?**

A non infectious, chronically recurring, papulosquamous disease, with combined cutaneous **inflammation** and **hyperproliferation**.

- **Does it have a definite cause?**

No.

G. psora, the itch

However, it is only itchy in 30%

- **Can you, briefly, explain the pathogenesis?**

A **genetic predisposition** makes the skin more liable to **inflammation** and **hyperproliferation** upon exposure to one of many **precipitating** factors.

- **Shall you mention some of such precipitating factors?**

- ❑ Trauma
- ❑ Infection
- ❑ Hormonal disturbances
- ❑ Sun exposure
- ❑ Drugs **e.g.** β-blockers, antimalarials, lithium
- ❑ Alcohol and cigarette smoking

What are its histopathological changes?

- **Parakeratosis**: retained nuclei in stratum corneum
- Regular **acanthosis**
- Neutrophil microabscesses
- Dilated dermal capillaries
- Upper dermal lymphocytic infiltrate

- **What is the main complaint?**

Disfigurement.

- **What are the clinical types?**

- ❑ According to **morphology**, we may have:
plaque, guttate, pustular.....
- ❑ According to **site**, we may have:
scalp, nail, genital.....

- **Which one is called “psoriasis vulgaris”?**

The **PLAQUE** type (chronic stationary):

Well-defined, glossy pink, covered with silvery, non-coherent scales (positive Auspitz’ sign)

Mostly on the elbows, knees, lower back, scalp symmetrical, variable in size.

- **What is guttate (eruptive) psoriasis?**

Numerous, small, round (**rain drop-like**), red **PAPULEs**, suddenly on the trunk followed by scaling. It usually affects children and adolescents often following streptococcal infection (tonsillitis).

L. gutta, drop

- **Who is Auspitz?**
Auspitz, H., German Physician, 1835-1896
- **Who is Köbner?**
Köbner, H., German Dermatologist, 1838-1904.

What is the “Auspitz’ sign”?

Small bleeding points occurring on psoriatic lesions, after removing scales (thin epidermis on long papillae).

- **What are the most common nail lesions?**
Pitting, oil drop, onycholysis, subungual keratosis

What is the Köbner’s phenomenon?

Development of skin lesions (e.g. psoriasis) at the site of physical trauma (e.g. scratching)

- **How can you treat psoriasis?**

□ General measures

- ✓ Reassurance and explanation
Patient must realise that there is no current curative treatment.
All modalities aim to suppress or make the disease more tolerable.
- ✓ Physical and mental rest

□ Topical measures

- ✓ Coal tar
- ✓ Dithranol
- ✓ Calcipotriol (vitamin D analogue)
- ✓ Topical steroids
- ✓ Topical retinoids e.g. tazarotene

□ Systemic measures

- ✓ Retinoids e.g. acitretin
- ✓ Methotrexate
- ✓ Cyclosporin
- ✓ Radiotherapy
 - UVB
 - PUVA (UVA + Psoralen)



Psoriasis; nail



Vulgaris



Guttate



Köbner's

Psoriasis

- **What is PRP?**
Pityriasis Rubra Pilaris, a rare papulosquamous disorder.
- **Which age groups are affected?**
 - Adults : acquired, self-limited (the majority of cases)
 - Children: familial
- **What is the primary defect?**
Epidermal hyperproliferation.
- **Is it due to vitamin A deficiency?**
NO.
- **How do the classic type present?**
 - Psoriasiform plaques: scaly, orange-red, with sharp borders & islands of free skin
 - Palmo-plantar keratoderma
 - Follicular hyperkeratosis
 - Diffuse scaling of the scalp
 - Nail dystrophy

- **What is pityriasis rosea?**

An acute, benign, self-limiting, papulosquamous disorder.

- **Do you know the exact aetiology?**

Nobody does!! {An infectious agent has been suspected, but not confirmed. It is not contagious. }

- **What is the “herald patch”?**

Its initial lesion: single, large, round **PLAQUE** with peripheral collarette of scales.

Subsequent lesions are: multiple, small, oval **macules**, mainly on the trunk (Christmas tree pattern), *sparing* face, palms and soles.



Herald patch



Pityriasis rosea

- **Is lichen planus a common disease?**

YES, relatively, constituting about 1% of dermatological consultations.

- **What is its most prominent symptom?**

Intense itching. Characteristically, *rubbing* rather than scratching, that may lead to the polished appearance of the papule.

- **Can you describe the classical skin lesions?**

Flat-topped, shiny, violaceous, polygonal (angulated) **papules**, 1-5 mm in diameter.

They occur mostly on the extremities, especially wrist flexors.

- **What are the “Wickham’s striae”?**

Lacy white network on the surface of skin lesions.

G. leichēn, a kind of plant growing on rocks
L. planus, flat

- **Does lichen planus affect the oral mucosa?**

YES, in up to 50% of cases.

- **...what about the nails?**

They are affected in about **10%** of cases, mostly as nail dystrophy.

- **Is it a systemic disease?**

NO. However, it might be associated with systemic disease e.g. hepatitis, malignancy.

- **What is the most serious complication?**

Squamous cell carcinoma, on top of oral ulcers.



Lichen planus

BLISTERING DISEASES*

• Can you classify vesiculo-bullous skin diseases?

- According to aetiology, we have:
 - ✓ Genetic e.g. epidermolysis bullosa**
 - ✓ Autoimmune e.g. pemphigus, pemphigoid, dermatitis herpetiformis (D.H.)
 - ✓ Infectious e.g. impetigo, herpes simplex, herpes zoster
 - ✓ Metabolic e.g. diabetic bullae, pellagra
 - ✓ Physical e.g. burns
 - ✓ Drugs e.g. tetracycline, sulfonamides
 - ✓ Others e.g. malignancy
- According to split-level we have:
 - ✓ Subcorneal e.g. impetigo
 - ✓ Intraepidermal e.g. acute eczema, herpes, pemphigus
 - ✓ Basilar e.g. epidermolysis bullosa simplex
 - ✓ Subepidermal e.g. D.H.,
- According to pathogenesis, we have:
 - ✓ Spongiotic e.g. eczema (page 36)
 - ✓ Acantholytic ♣ e.g. pemphigus
 - ✓ Cytolytic e.g. herpes
 - ✓ Dissolved basement membrane e.g. bullous pemphigoid
 - ✓ Damaged dermal papillae e.g. D.H.

• Can you mention main features of pemphigus vulgaris, bullous pemphigoid, D.H.?

	Pemphigus vulgaris	Bullous pemphigoid	D.H.
Age (in years)	40-50	60-80	20-40
General condition	Poor	Good	Itchy
Skin sites	Trunk, face, scalp	Flexures	Elbow, knees, buttocks
Blister nature	Flaccid	Tense, blood-filled	Grouped, small excoriated
Immunoglobulins			
Circulating	IgG	IgG	IgG
Fixed	IgG	IgG	IgA
Treatment	Steroids	Steroids	Dapsone
	Immunosuppressants	Immunosuppressants	Gluten-free diet



*Both vesicles and bullae are known as blisters.

**genetically determined tendency to develop blisters at different levels, spontaneous or post-traumatic

♣ACANTHOLYSIS: loss of epidermal cell coherence

RHEUMATIC DISEASES WITH SKIN MANIFESTATIONS¹

G. *rheuma*, flux i.e. continued change
Rheumatism is an indefinite term applied to various conditions with symptoms related to the musculo-skeletal system.

- **What do you mean by rheumatic diseases?**

Any of a variety of disorders marked by inflammation, degeneration, or metabolic derangement of the connective tissue structures of the body. They are autoimmune, in origin, affecting genetically-predisposed individuals.

They are classified together on the basis of the widespread fibrinoid degeneration of collagen fibers.

- **What are their cardinal features?**

Chronic inflammation of connective tissue, with **dermal atrophy** or **sclerosis**, arthritis and other organ abnormalities. Formation of auto-antibodies against normal tissue components is striking.

- **Can you tell me some examples?**

- ❑ Lupus erythematosus (L.E.)
- ❑ Scleroderma
- ❑ Dermatomyositis

- **What is L.E.?**

A heterogeneous spectrum of connective tissue disorders, including:

- ❑ Chronic discoid L.E. (**DLE**): almost always limited to skin
- ❑ Subacute L.E. : may involve systemic organs
- ❑ Acute systemic L.E. (**SLE**) : potentially fatal, multisystem disease overshadows skin lesions



- **Would you please, tell me the striking features of DLE?**

Well-defined, disc-like **PLAQUES**, with scaling, hyperkeratosis and follicular plugging. Mostly on the face and scalp. Central healing may be followed by either atrophy or depigmentation.

- **What are the main treatment lines of DLE?**

- ❑ Sunscreens
- ❑ Topical steroids
- ❑ Antimalarials

What are the most important skin signs of SLE?

- Malar flush
- Discoid plaques
- Photosensitivity

*You should have studied the other signs in internal medicine!
Didn't you?!!!!*

- **What is scleroderma?**

A group of connective tissue diseases involving microvasculature and connective tissue ending with fibrosis.

- **What are its types?**

- localised "morphea"**: smooth, sclerotic (hard) **plaque**, with ivory (whitish) centre and lilac (violaceous) border.
- generalised**: involving visceral organs as well.

- **What is dermatomyositis?**

A group of connective tissue diseases involving skin and skeletal muscle. **Visceral malignancy** may occur.

- **What are the main cutaneous features?**

- ❑ **Gotttron's papules**: erythematous/purple flat papules on the back of the interphalangeal joints.
- ❑ **Gotttron's sign**: symmetrical violaceous erythema & oedema over knuckles, elbows & knees.
- ❑ **Heliotrope erythema**: lilac-colored, periorbital oedema (which has been likened to the heliotrope flower)
- ❑ **Periungual telangiectasia**
- ❑ **Photodistributed violaceous erythema**

¹ The term "**collagen diseases**" should never be used as a synonym, as collagen tissue is only secondarily involved in such diseases.

PIGMENTARY DISORDERS

- Do you know the cause of **NORMAL** skin colour?

A mixture of pigments

- ❑ **Melanin***, brown: in epidermis
- ❑ **Oxyhaemoglobin**, red: in arterioles and capillaries
- ❑ **Deoxyhaemoglobin**, blue: in venules
- ❑ **Carotene**, yellow: in dermal connective tissue

Is it common?

YES, most of us have got some type during life time.

What is “**depigmentation**”?

Abnormal pigmentation, may be:

- hypo / hyper ¶
- epidermal / dermal
- melanin / non-melanin
- genetic / acquired

- What is the mechanism of **RACIAL** differences in pigmentation?

- Different melanin **type**

- Variable melanin **amount**

What is its cause?

- ↓ or ↑ normal pigments
- deposition of abnormal pigment

- Would you mention **SOME** causes of depigmentation?

	HYPOPIGMENTATION			HYPERPIGMENTATION				
	EPIDERMAL		DERMAL	EPIDERMAL		DERMAL		
	↓ melanocytes	↓ melanin		↑ melanocytes	↑ melanin	↑ melanocytes	↑ melanin	non-melanin
Genetic	vitiligo	albinism	anemic nevus	lentigens	freckles	blue nevus	incontinentia pigmenti	-
Chemicals	Paraphenols	hydroquinone	-	-	psoralens	-	-	minocycline
Physical	burns	dermabrasio	-	UVR	UVR	-	-	-
Endocrinal	-	hypopituitarism	-	-	Addison's	-	-	-
Nutritional	↓ vit. B12	kwashiorkor	-	-	pellagra	-	-	ch. deficiency
Inflammatory	pinta	pityriasis alba	-	-	postinflam.	-	-	-
Others	-	-	anemia, oedema	-	-	-	-	-

- What happens in vitiligo?

Complete loss of melanocytes in the affected area.

L. vitium, an ugly mark

Why...?

Unknown. However more than one theory is suggested e.g. autoimmune, genetic, ...

- How common is it?

It affects about 1% of the population. Both sexes, all races & age groups are affected

What is the role of trauma and sunburn?

They **might** precipitate the disease, in predisposed individuals.

- How does it present?

Non-itchy, sharply-defined, milky white

MACULES/PATCHES,

mainly acrofacial. Course is unpredictable.

- How would you treat it?

- ❑ **Medical:**
 - PUVA, 1st choice
 - Topical steroids
 - Camouflage / depigmentation
- ❑ **Surgical:**
 - Split-skin graft, 1st choice #
 - Melanocyte culture
 - Minigraft
 - Micropigmentation



Vitiligo

* Melanin is the major component of skin colour, it is synthesised in cytoplasmic organelles “melanosomes”, within melanocytes,

¶ Leukoderma: white skin (↓ melanin), Melanoderma: brown skin (↑ melanin, in epidermis)

Only for localised, stable, medically-unresponsive cases

GENODERMATOSES

- **What are genodermatoses?**
Skin disorders of genetic origin.

Can you give examples?
-Ichthyosis
-Xeroderma pigmentosum
-Epidermolysis bullosa, p. 41

- **What is ichthyosis?**
Heterogeneous group of disorders characterised by excessive skin scaling.
 - ❑ **Hereditary, e.g.** Ichthyosis vulgaris
X-linked ichthyosis
 - ❑ **Acquired, e.g.** Malignancy
Leprosy

G. *ichthys*, fish
osis, condition

- Can you differentiate between ichthyosis vulgaris and X-linked ichthyosis?

	Ichthyosis vulgaris	X-linked ichthyosis
Onset	Childhood	birth, infancy
Site	extensors, <i>sparing flexures</i>	face, neck, trunk, flexures
Scales	fine, white	coarse, brown
Associations	atopy	cataract, cryptorchidism
Granular layer	decreased	normal
Molecular defect	↓ filaggrin	↓ steroid sulfatase

- **How can you treat?**
 - ❑ Acquired: treat the cause
 - ❑ Hereditary: - **Topical**: emollients e.g. urea, lactic acid
- **Systemic**: retinoids
- **What is “collodion baby”?**
Most common presentation of different types of ichthyosis in the newborn.
The child is encased in a transparent shiny, tough, membranous covering . It breaks up within 2 weeks.
- **What is “Harlequin foetus”?**
Rare form of ichthyosis in the newborn. Child is covered with very thick plates of stratum corneum.
- **What is xeroderma pigmentosum?**
A group of rare, fatal, autosomal recessive disorders, with defective repair of UV-damaged DNA.
- **What are the clinical lesions detected in such cases?**
 - ❑ Infants: photosensitivity
 - ❑ Children: freckles, solar keratoses
 - ❑ Adults: squamous cell carcinoma, basal cell carcinoma, malignant melanoma.
- **How would you manage?**
 - ❑ **Preventive measures**: prenatal diagnosis, if parents have already had one affected child.
 - ❑ **Active measures** : strict sunlight avoidance, follow-up



HAIR DISORDERS

• Can you briefly describe the hair follicle?

An epidermal invagination extending deep into the dermis

Hair **shaft** is the long slender filament in the center of the follicle, surrounded by internal & external root sheath, glassy membrane and connective tissue sheath. Hair **bulb** is the terminal expanded region of the follicle in which hair is rooted. It is deeply indented by the **dermal papilla**.

What is the scalp hair growth rate?
1 mm / 3 days

• Would you tell me the types of hair?

- ☐ Lanugo: fine, long, unmedullated, covering foetus
- ☐ Vellous: fine, short, unmedullated, covering body
- ☐ Terminal: coarse, long, medullated, covering scalp, axilla,...

What is the glabrous skin?
Hair-free skin: palms, soles, lips
glans, labia minora

• Do you know the hair growth cycle?

Each follicle passes through the following cycle:

- ☐ **Anagen phase**: active, for 1000 days
- ☐ **Catagen phase**: convertive, for 10 days
- ☐ **Telogen phase**: resting, for 100 days, after which, hair is shed

• What are the types of hair abnormalities?

- ☐ **Alopecia**: loss of hair
- ☐ **Hirsutism**: excess growth of terminal hair in a ♂ pattern (in ♀ one)
- ☐ **Hypertrichosis**: excess growth of terminal hair **NOT** in a ♂ pattern
- ☐ **Hair shaft abnormalities**

• What are the types of alopecia?

- ☐ **Cicatricial***
- ☐ **Non-cicatricial****

G. alōpex, a fox

• What is alopecia areata?

Uniform, non-scaly totally bald **patch**, mostly on scalp.
Exclamation mark hair are often noted at the periphery.
{It is pathognomonic. (These are broken-off about 4mm from the scalp surface, thinner and less pigmented proximally)}.
Nail pitting or ridging may occur, especially in severe chronic cases.

• How can you treat it?

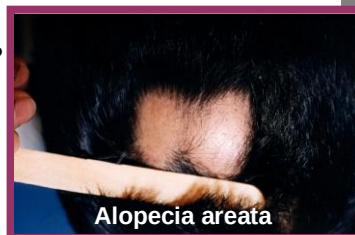
- ☐ Immunomodulators: Steroids, Anthralin, PUVA
- ☐ Biologic response modifiers: minoxidil

• What is alopecia totalis?

Loss of all the scalp hair.

• What is alopecia universalis?

Loss of all the body hair.



What are the causes of cicatricial alopecia?

- ☐ **Developmental/hereditary**
 - Epidermal nevi
- ☐ **Inflammatory**
 - Acute**
 - Viral: herpes simplex, zoster
 - Bacterial: lupus vulgaris deep folliculitis
 - Fungal: kerion, favus
 - Leishmaniasis
 - Chronic**
 - DLE, LP, morphea
- ☐ **Physical**
 - Trauma
 - Radiation
- ☐ **Neoplastic**
 - Basal or squamous cell carcinoma

What are the causes of non-cicatricial alopecia?

- ☐ **Hereditary**
- ☐ **Androgenetic**
- ☐ **Alopecia areata**
- ☐ **Traumatic**
 - Trichotillomania
 - Traction
- ☐ **Systemic disease**
 - Endocrinopathy
 - Stress:
 - (Anagen effloviom -post-partum)
 - (Telogen effloviom -post-febrile)
 - Drugs:
 - (Telogen effloviom) -post-operative
 - cytotoxic
 - anticoagulants
 - antithyroid
 - Deficiency states:
 - protein malnutrition
 - iron deficiency
 - syphilis

*hair follicles are destroyed or malformed, replaced by fibrous tissue i.e. no longer can produce hair

**due to either transformation of terminal to vellous hair follicles or disordered hair cycle

NAIL DISORDERS

- **Can you describe, briefly, the nail structure?**

The nail **matrix**, gives rise to nail **plate**. The plate rests on nail **bed** and is surrounded on three sides by nail **folds**.

- **What are the functions of nails?**

- ☐ Protect the ends of fingers and toes.
- ☐ Increase tactile sensitivity (counter pressure over distal pulp).
- ☐ Relieve itching
- ☐ Extend beauty.

Is it mainly composed of calcium?
NO, it is made of hard keratin.
 Calcium accounts for only a small %.

What is the nail growth rate?
Finger nails: 1 mm/10 days
Toe nails : 1 mm/30 days

- **Can you mention some of the nail disorders?**

Nail disorders can be classified in more than one way:

- ☐ According to the onset of the lesion: congenital or acquired
- ☐ According to the nail anatomical site affected (matrix, bed, plate, fold)
- ☐ Whether it is a localized nail disorder or a part of a multi-systemic disorder

Here, you can find some **EXAMPLES** of nail disorders, you may see at your clinic:

Affected site: change	Disorder: causes
Nail plate: red distal 1/3, white proximal 2/3	Terry nails: congestive heart failure, cirrhosis
Nail plate: red distal 1/2, white proximal 1/2	Half-and-Half nails: chronic renal failure
Nail bed: brown red streaks	Splinter hemorrhages: trauma (distal), bacterial endocarditis (middle)
Nail plate/bed: brown	Addison's disease, hemochromatosis, gold, arsenic, melanoma
Nail plate: blue	Blue nails: antimalarial, minocyclins, hemochromatosis, Wilson's dis.
Nail plate: yellow, dystrophic	Yellow nail syndrome: lymphatic abnormality, systemic illness/cancer
Nail plate: Partially separation from nail bed	Onycholysis: psoriasis
Nail plate: Pitting	Psoriasis, alopecia areata
Nail plate: soft, thin; hence concave "spoon"	Koilonychia: iron-deficiency anemia, plummer-Vinson syndrome
Nail plate: horizontal depressions	Peau's lines: transient nail growth arrest e.g. fever, shock, infarction
Nail plate: Cuticle extending over the plate	Pterygium: Lichen planus
Nail fold: telangiectasia	Immune complex deposits: Dermatomyositis, Systemic LE
Nail fold: Inflammation	Paronychia: bacterial, candidal

MISCELLANEOUS

- **What are chilblains?**

Painful-itchy, purple/pink, inflammatory swelling, on the fingers, toes, ears. It may represent vasculitis due to cold weather
 Treatment includes warm housing and clothing beside calcium channel blockers, nicotinamide and sympathectomy.

- **What is miliaria?**

Sweat retention secondary to sweat duct obstruction. Presentation depends on level of obstruction:

	Site	Inflammatory response	Lesion	Aetiology
Miliaria crystallina	Most superficial	Minimal to non	Asymptomatic tiny clear vesicles	Sunburn
Miliaria rubra	Intraepidermal	Moderate	Tiny erythematous papulovesicles	Cloths friction
Miliaria profunda	Deepest 'DEJ'	Severe	Larger erythematous papules	Tropics

N.B. Miliaria pustulosa results from secondary bacterial infection and is characterised by flaccid pustules

Treatment involves: -move to a cooler climate (AC) -avoid cloths, which prevent sweat evaporation -give antibiotics in miliaria pustulosa

NEOPLASMS

• Shall you mention some of the skin tumors?

	Benign	Premalignant	Malignant
Epidermal	Seborrheic keratosis Skin tag Miliun Melanocytic naevus	Actinic keratosis	Basal cell carcinoma Squamous cell carcinoma Malignant melanoma
Dermal	Haemangioma Lipoma		Kaposi's sarcoma Lymphoma

• What is seborrheic keratosis?

A common, benign, epidermal tumor. Multiple, round/oval, pigmented, papulo-nodules, with well-defined edge and stuck-on appearance. Mostly on face/trunk, usually after age of 50.

• What is skin tag (acrochordon)?

Common, benign, epidermal tumor. Soft, skin-colored, pedunculated, papules, mostly on neck/flexures, in middle aged & elderly

• What is a naevus?

A birthmark.

• What is a mole?

A common name of melanocytic naevus. It is derived from "naevus" cells

• Can you mention its types?

- ☐ **CONGENITAL:** since birth, > 1 cm, brown/black, protuberant hairy mostly on the trunk.
- ☐ **ACQUIRED:**
 - **Junctional:** macule, mid/dark brown mostly on limbs.
 - **Compound:** dome nodule, color is more even but less pigmented, smooth surface, anywhere.
 - **Intraepidermal:** dome nodule, skin-colored, mostly on face/neck.
 - **Others:**
 - ✓ **Spitz:** single, red nodule, up to 1 cm in diameter on children faces
 - ✓ **Blue:** single, slate gray blue nodule on extremities due to accumulation of dermal melanocytes
 - ✓ **Dysplastic:** irregular edge, variable color
 - ✓ **Halo:** trunkal nevus surrounded by depigmented area

• Which ones may turn malignant?

- Congenital
- Dysplastic

• What is lipoma?

Multiple, soft, subcutaneous mass
Trunk, neck, upper extremities

• What is actinic keratosis?

A common precancerous tumor in fair complexioned persons affecting mainly the sun-exposed areas as multiple flat rough pigmented patches with adherent scales.



• What is the most important single cause of skin malignancy?

Sun exposure.

• Which is the commonest skin cancer?

Basal cell carcinoma.

• What are its features?

It has several variants, e.g.

- ☐ **Nodulo-ulcerative:** the most common. Glistening, translucent, skin-colored papulo-nodule that enlarges slowly with central necrosis and ulcer formation (rolled pearly well-defined edge covered with an adherent crust)
- ☐ **Morpheic**
- ☐ **Superficial**
- ☐ **Pigmented**

• What is its most important complication?

Local invasion & destruction
Metastases are too rare

• What is squamous cell carcinoma?

A common skin cancer that arise either de novo or on top of solar keratosis or leukoplakia as rapidly growing nodule soon develops a central crusted ulcer with an indurated base, raised irregular, everted edge mostly on sun-exposed areas (face).

• What is the fastest increasing cancer in western countries?

Malignant melanoma

It is more common in those with fair complexion, exposed to sunlight for short intense periods.
People with past or family histories are more susceptible.

• What are its criteria?

- ☐ **Asymmetry**
- ☐ **Border irregularity**
- ☐ **Color variegation**
- ☐ **Diameter > 0.5 cm**
- ☐ **Elevation irregularity**

• What are its types?

- ☐ **Lentigo maligna**
- ☐ **Superficial spreading**
- ☐ **Acrall lentiginous**
- ☐ **Nodular**

SEXUALLY TRANSMITTED DISEASES

“Venereal diseases”

L. *Venus*, the goddess of love
To indicate the relation to sexual intercourse

- **What do you mean by sexually transmitted diseases (STDs)?**
Infectious diseases transmitted only or mainly by means of sexual relations.
- **How are STDs classified?**
 - ❑ ***Etiological*** classification; according to: causative agents
 - ❑ ***Syndromic*** classification; according to: consistent groups of symptoms and signs
- **Can you mention some of the causative agents of STDs?**

Viruses		
<u>H</u> erpes <u>s</u> implex <u>v</u> irus	HSV-1,2	Genital herpes
<u>H</u> uman <u>p</u> apilloma <u>v</u> irus	HPV	Genital warts
Pox virus		Molluscum contagiosum
<u>H</u> uman <u>I</u> mmunodeficiency <u>V</u> irus	HIV-1,2	AIDS
Bacteria		
<u>N</u> eisseria <u>g</u> onorrhoea		Gonorrhoea
<u>T</u> reponema <u>p</u> allidum		Syphilis
<u>H</u> aemophilus <u>d</u> ucrei		Chancroid
<u>C</u> alymmatobacterium <u>g</u> ranulomatis		Granuloma inguinal
<u>U</u> reaplasma <u>u</u> realyticum		Urethritis
<u>C</u> hlamydia <u>t</u> rachomatis		
Serotypes <i>D-K</i>		Urethritis
<i>L1-3</i>		Lymphogranuloma venereum
<u>G</u> ardenerella <u>v</u> aginalis		Bacterial vaginosis
Fungi		
<u>C</u> andida <u>a</u> lbicans		Candidiasis
Protozoa		
<u>T</u> richomonas <u>v</u> aginalis		Trichomoniasis
Ectoparasites		
<u>P</u> hthirus <u>p</u> ubis		Pediculosis pubis
<u>S</u> arcoptes <u>s</u> cabiei		Scabies

What is the fastest growing STD?

What is the commonest STD?

- **Can you mention some of the sexually transmitted SYNDROMES?**
 - ❑ Urethral discharge syndrome
 - ❑ Ulcer/node syndrome
 - ❑ Vaginal discharge syndrome (in ♀)
 - ❑ AIDS
- **Which is more practical, the etiologic or the syndromic classification?**
The syndromic. According to the WHO and CDC, it is not easy to define exactly the causative agent using the current diagnostic methods which are often unavailable everywhere, expensive and/or requiring sophisticated equipment and well-trained personnel.

- **How to treat an STD?** *general principles applied to all*

- ❑ Eradication of the causative agent
- ❑ Education
- ❑ Contact tracing & treatment (partner's notification)
- ❑ Full screening for STDs
- ❑ Follow-up, in certain cases

Single-dose, oral regimen is preferred whenever possible

URETHRAL DISCHARGE

- What are the causes of urethral discharge in ♂?

- Physiological
 - Semen
 - Prosemen
 - prostatorrhoea
- Pathological
 - Urethritis

- How can you classify urethritis?

- Gonococcal
- Non-Gonococcal (NGU)

- How can you diagnose a case of urethritis?

- History: dysuria; burning or itching along the course of urethra
- Exam : urethral discharge, meatal inflammation
- Lab : **excess neutrophils** {5 or more/oil immersion field (x100)} **most important**
 - in a Gram-stained smear of urethral discharge
 - or in the sediment of “first-voided” urine

- Is it recommended to test for microorganisms other than gonococci?

NO. This is impractical and poorly cost-effective.

- Can the discharge’s macroscopic features differentiate between gonorrhea and NGU?
NO.

- What is the causative agent of gonococcal urethritis?

Neisseria gonorrhoea (gonococci):

- Gram-negative (pink-colored, do not retain the dye)
- Diplococci (occurring in pairs)
- Kidney-shaped
- Intracellular, *mostly* (within neutrophils)
- Humans are the only natural host

G. gonē, seed
rhoia, a flow

- What is the size of the problem?

About **200** million new cases/year are notified to the WHO. However, real number is certainly higher

- What is the spectrum of gonococcal infection?

- **LOCALIZED** infections, affecting a wide range of **columnar** mucous membranes:
 - Urethra
 - Genital glands
 - Uterine cervix, fallopian tubes
 - Anal canal, distal rectum
 - Conjunctiva
 - Pharynx
- **DISSEMINATED**, blood-born infections (1% of cases)

• **Do you know the anatomy of male urethra?**

Its length is about 20cm, and can be divided into 3 successive parts:

- ❑ **Prostatic** : 3 cm, lined by transitional epithelium *posterior* urethra
- ❑ **Membranous**: 2 cm, „ „ columnar „ }
- ❑ **Spongy** : 15 cm, „ „ „ „ } *anterior* urethra

except for the outermost part (fossa navicularis)

• **Can you name the urethral glands?**

- ❑ **Littre's** “periurethral” : draining mucous into the anterior urethra
- ❑ **Cowper's** “bulbourethral” : „ „ „ „ „ „ „ „

N.B. **Tyson's** glands are modified sebaceous glands opening directly on the inner surface of the prepuce.

• **What is the most common site of primary infection in ♂?**

Urethra, leading to acute anterior urethritis,

- Presenting with* {
- ❑ **Dysuria**
 - ❑ Urethral **discharge**; profuse, purulent, yellow (staining the underwear)
starting **2-5** days after sexual contact (**IP**)
peaks within 2 weeks
resolves within few weeks, if not treated
 - ❑ Erythema and oedema of urethral meatus

5-15% of cases are *asymptomatic*

What is the “Two-glass test of urine”?

An office test to determine roughly, the extent of the condition. Ask patient to pass urine into two separate glasses.

- ❑ Cloudiness of the **1st** glass means **anterior** urethritis.
- ❑ Cloudiness of **both** glasses indicates involvement of the **posterior** urethra as well.

• **What is the most common site of primary infection in ♀?**

Cervix uteri (columnar-lined endocervix)

the condition may **be asymptomatic** (>50%)

or present with non-specific symptoms e.g. vaginal discharge, dyspareunia, intermenstrual uterine bleeding, low backache

Exam: mucopurulent cervical discharge, erythema, and edema

• **What are the most USEFUL diagnostic methods?**

- ❑ Gram-stained smear: **98%** sensitive in acute anterior urethritis (in ♂)
- ❑ Culture on a selective medium e.g. modified *Thayer-Martin* medium

essential in:

- ♀ (specimen taken from endocervix)
- ♂ - Urethral infections
- Acute cases (with -ve smear i.e. 2%)
- Chronic/asymptomatic cases
- Rectal or pharyngeal infections

What is the advantage of the recently-developed gene amplification methods e.g. PCR?

- ❑ Most sensitive
- ❑ Easy specimen collection
(you can use urine to detect gonococci !!!!)

What are their disadvantages?

- ❑ Longer time to get results (vs. *smear*)
- ❑ Cannot detect antibiotic sensitivity (vs. *culture*)

منتصب القامة أمشي

مرفوع الهامة أمشي

في كفي قطفة زيتون

و على كتفي نعشي

• What are the complications of primary gonococcal infections?

□ **LOCAL:**

◆ ♂ **RARE** now

- Posterior urethritis frequency, urgency, terminal haematuria
- Prostatitis
- Seminal vesiculitis
- Cowperitis
- Tysonitis
- Epididymitis unilateral scrotal pain and swelling
- Lymphangitis
- Penile edema
- Urethral stricture & periurethral abscess

◆ ♀ **more COMMON**

- **P**elvic **I**nflammatory **D**isease (Acute salpingitis) {*the most common, 10-20%*}
- Bartholin's abscess

□ **SYSTEMIC** (Bacteraemia; disseminated gonococcal infection): *more common in ♀*

- ◆ **Dermatitis-arthritis** syndrome mostly
- ◆ Endocarditis rare
- ◆ Meningitis rare

• What is the role of penicillin in the current management of gonorrhoea?

Traditionally, penicillin (crystalline) was the mainstay of gonococcal control.

However, with increasing incidence of resistant strains, more effective drugs have replaced it.

It should not be used **except** after testing for antibiotic sensitivity, to be sure that penicillin is still effective.

• How can you eradicate gonococcal infections?

Drug of choice		Alternative	
Ceftriaxone	250mg I.M., once	Ciprofloxacin	500mg oral, once
<i>for</i> uncomplicated urethral, cervical, rectal, or pharyngeal infections		(contraindicated in pregnancy)	

Tetracyclines, for 7 days, should be added to eradicate concomitant *chlamydial* infections.

• What is the most difficult gonococcal infection to eradicate?

Pharyngitis

• What to do for other types of gonococcal infections?

Consult.

• Is it essential to test for cure after using the above-mentioned regimen?

NO, because of the extremely high cure rates.

إن لم تستح، فافعل ما شئت

- **How common is the NGU?**

It is the most common STD in western countries. However, the number is declining in the last few years.

- **What are its main causes?**

- ❑ *C. trachomatis* 60%
- ❑ *U. urealyticum* 20%
- ❑ Others 20%
e.g. - *T. vaginalis*
- HSV

- **What is its clinical presentation?**

- ❑ **Dysuria**, irritation along the extension of the urethra
- ❑ **Discharge**; mostly mucoid
- ❑ Asymptomatic; 30%

- **What are the complications?**

- ❑ Persistence
- ❑ Epididymitis, prostatitis
- ❑ Reiter's syndrome

- **How can you treat NGU?**

Drug of choice	Alternative
- Doxycycline 100 mg/12 hours for 7 days	- Erythromycin
OR - Oxytetracycline 500 mg/6 hours for 7 days	OR - Azithromycin

بسم الله الرحمن الرحيم

ومن الناس من يعجبك قوله في الحياة الدنيا، ويشهد الله على ما في قلبه، وهو ألد الخصام. وإذا تولى سعي في الأرض لفسد فيها ويهلك الحرث والنسل، والله لا يحب الفساد. وإذا قيل له اتق الله أخذته العزة بالإثم، فحسبه جهنم، ولبئس المهادر.

ULCER / NODE

- What is the “Great pox”?

It's **syphilis** (vs. smallpox)

- Is it a rare disease?

NO. It was decreasing till the late 1980s, when it started to rise markedly (drug abuse, prostitution).
About **50** million cases are reported annually, WHO.

- What is the causative agent?

The spirochete: ***Treponema pallidum***.

It is a spiral, motile bacterium.

It **CANNOT** be illustrated by ordinary light microscopy.

How can you identify it?

- ☐ **Dark ground** microscopy
- ☐ Silver staining
- ☐ Immunofluorescent staining
- ☐ Electron microscopy

Why?

Too small !!

Is it cultivable?

NOT on ordinary media.
However it can be grown
in rabbit's testicles.

- What would you do to avoid catching infection during practice?

Intact rubber gloves are enough.

- How to disinfect?

Washing with soap and water (the organism is very fragile)

- How can you classify syphilis?

- ☐ According to the duration of infection:

- . early: 1st two years full of *T. pallidum*
- . late : afterwards few or no *T. pallidum*

- ☐ According to the beginning of infection:

- . acquired (postnatal)
- . congenital (prenatal)

- How is it transmitted?

- ☐ **Acquired**

- Sexual relations {it **CANNOT** penetrate intact skin or mucosa; abrasions must be present}
- Accidental {e.g. during transfusion, surgery.....}

- ☐ **Congenital**

- Transplacental

- How long is the incubation period of syphilis?

9-90 days

Why is it so variable?

Depending on the quantity of the invading spirochetes

- How long is its infectious period?

- ☐ Definite : -Early syphilis (i.e. 1st two years)
- ☐ Possible: -Incubation period
- Late syphilis

- **What is the natural history of syphilitic infection?** (i.e. What would happen if not treated?)
 - ❑ **Primary stage** : Spirochetes invade, multiply and stimulate **LOCAL** tissue reaction with:
 - Plasma cells/lymphocyte infiltration
 - Endarteritis obliterans
 - Local necrosis in the form of **chancre**
 - ❑ **Secondary stage**: Spirochetemia (blood dissemination) with **SYSTEMIC** manifestations
 - ❑ **Latent stage** : A state of balance between the spirochetes and the host immunity
 - may** -remain as such **1/3** of cases
 - or may** -spontaneously cure **1/3** of cases
 - or may** -progress to tertiary stage **1/3** of cases
 - ❑ **Tertiary stage** : -**Benign**
 - Skin
 - Mucous membranes
 - Malignant**
 - Cardiovascular
 - Neurological

Is syphilis fatal?
YES, in 15% of untreated cases.

- **What is the “Hunterian chancre”?**

The classical primary syphilitic lesion, representing **ONLY 50%** of cases. It consists of an eroded papule (**ulcer**) which is self-limited (within 2-6 weeks) leaving thin scar.

Compared to soft sore:

	Hard sore (chancre)	Soft sore (chancroid)
IP	-9-90 days	-3-5 days
Number	-single	-multiple
Pain	-painless, tenderless	-painful, tender
Shape	-round	-irregular
Border	-well-defined	-well-defined, ragged, undermined
Floor	-clean, red, covered with yellowish grey scab	-dirty, covered with greyish yellow necrotic exudate
Base	-hard (indurated)	-soft
Site	-♂: coronal sulcus, glans penis -♀: labia, clitoris, cervix	-♂: prepuce, frenulum, coronal sulcus -♀: fourchette, labia, vestibule, clitoris
Bleeding	-not easy	-easy
Oozing	-serum full of spirochetes	-seropus
Lymphadenopathy*	-bilateral inguinal within 1-2 weeks -painless, tenderless, large, discrete, firm, rubbery, freely mobile, overlying skin is normal	-uni- or bilateral inguinal within 1 week -painful, tender, large, matted, may fluctuate (50% of cases) -overlying skin is erythematous
Dark ground	- +ve	- -ve
Treatment	-Penicillin (benzathine)	-Erythromycin

* **bubo**: inflammatory swelling of one or more lymph nodes in the groins.

- **How could chancre be atypical?**

Painful, multiple, extragenital,....

Can you remember some causes of painful chancre?

- ❑ Secondary pyogenic infection
- ❑ Mixed infection with **H. ducreyi**
- ❑ Subungual

Do you know some causes of multiple chancre?

- ❑ Reinfection within the 1st two weeks (before development of immunity)
- ❑

- What is **syphilis d'emblée**?

Deep inoculation of *T. pallidum* results in syphilitic infection with **absent chancre** e.g. infected needle puncture

Fr. **d'emblée**, right away

- What is **chancre redux**?

New **chancre** developing on the scar of an old chancre.

- What is **pseudochancre redux**?

Gumma developing on the scar of an old chancre.

every genital ulcer should be considered **syphilitic**, till proved otherwise

- What is the differential diagnosis of the Hunterian chancre?

- ❑ **Sexually transmitted**

chancroid, **g**ranuloma inguinale, **l**ymphogranuloma venerum, **h**erpes progenitalis, **s**cabies

- ❑ **Non-sexually transmitted**

fixed drug eruption, **t**rauma (secondarily infected), **T.B.**, **n**eoplasm

- What is the most reliable method to confirm diagnosis of primary syphilis?

Dark-ground microscopy

Which material can be examined in this test?

- ❑ **Oozing serum**, from ulcer
- ❑ **Aspirate**, from enlarged lymph nodes

Advantages : rapid, specific

Disadvantages: not easily available, needs well-trained personnel

- When does the secondary stage start?

Six weeks after the onset of chancre,

(whether chancre has disappeared, OR not)

- What are the clinical presentations of 2nd stage?

According to the frequency of occurrence:

- ❑ **Syphilides**

- ❑ Generalized lymphadenopathy
 - ❑ Constitutional manifestations
 - ❑ Other organ affection

What is its differential diagnosis?

It is the great imitator, **may simulate**: **m**easles, **d**rug eruption, **p**ityriasis rosea, **l**ichen planus, **p**soriasis, ...

What are the main characters of syphilides?

Widespread, symmetrical, non-itchy (usually)

- What are the most commonly affected sites?

Soles, palms, trunk, genitalia, mucosa

- What are “syphilides”?

These are **syphilitic rash**:

- Macular
- Papular
- Papulosquamous
- Pustular
- Alopecia
- Mucosal lesions

What are condylomata lata?

Modified **papules** with: grayish white **color**, flat, smooth, **surface**, broad, indurated **base**.

Location: **intertriginous** areas e.g. genitalia, perianal.

More common in ♀

The **most infectious** syphilitic lesion

What are the types of 2nd stage alopecia?

- Localized moth-eaten **non-scarring**
- Diffuse telogen effluvium

What do you know about mucous **patches**?

Painless, shallow, grayish white **erosions** of mucous membranes e.g. lips, tongue, urethra

- **How to confirm diagnosis of 2nd stage?**

Through the **STS** (Serological Tests for Syphilis)
They are **100% +ve** (except in *AIDS*)

- **What are the STS?**

Examining the patient's **serum** for the presence of **antibodies** (Ig) directed against *T. pallidum*.

- **What are the types of STS?**

- **Non-Treponemal**

- **Screening:** Testing the presence of reagin antibodies in patient's serum.
 - **Highly sensitive:** not specific i.e. many false +ve
 - e.g. – **VDRL:** Venereal Disease Research Laboratory
 - **RPR:** Rapid Plasma Regain

- **Treponemal**

- **Confirmatory:** Testing the presence of specific anti-treponemal antibodies in patient's serum.
 - **Highly specific**
 - e.g. – **TPI:** Treponema Pallidum Immobilisation
 - **TPHA:** Treponema Pallidum HaemAgglutination
 - **FTA-Abs:** Fluorescent Treponemal Antibody-Absorption

- **What is the relation between syphilis and AIDS?**

HIV infection may affect syphilis:

- Alter clinical manifestations
 - Alter the STS (become –ve, when should be +ve)
 - Result in mixed infection
 - Decrease latency period
 - Decrease efficacy of antibiotics

Syphilis may facilitate acquisition of HIV infection (*via chancre*)

اسْتَعْمَلَ النَّبِيُّ صَلَّى اللَّهُ عَلَيْهِ وَسَلَّمَ رَجُلًا عَلَى الصَّدَقَةِ فَلَمَّا قَدِمَ قَالَ هَذَا لَكُمْ وَهَذَا أُهْدِيَ لِي قَالَ فَهَلَّا جَلَسَ فِي بَيْتِ أَبِيهِ أَوْ
بَيْتِ أُمِّهِ فَيَنْظُرُ يُهْدَى لَهُ أَمَّ لَا وَالَّذِي نَفْسِي بِيَدِهِ لَا يَأْخُذُ أَحَدٌ مِنْهُ شَيْئًا إِلَّا جَاءَ بِهِ يَوْمَ الْقِيَامَةِ يَحْمِلُهُ عَلَى رَقَبَتِهِ إِنْ كَانَ
بَعِيرًا لَهُ رِغَاءٌ أَوْ بَقْرَةً لَهَا خَوَازٍ أَوْ شَاةٌ تَيْعَرُ ثُمَّ رَفَعَ يَدَهُ حَتَّى رَأَيْنَا عُفْرَةَ إِبْطِيهِ اللَّهُمَّ هَلْ بَلَغْتَ اللَّهُمَّ هَلْ بَلَغْتَ ثَلَاثًا.

• Shall you mention the common causes of Tropical GENITAL ULCERS♠?

	Chancroid	Granuloma inguina	Lymphogranuloma veneru
Cause	- <i>H. ducrei</i>	- <i>C. granulomatis</i>	- <i>C. trachomatis</i> ; <i>L</i> ₁₋₃
IP	-3-5 days	-8-80 days	-7-28 days
Clinically	-starts as a papulopustule the ULCER is painful, tender, multiple, shallow, soft, undermined, ragged edge, bleeds easily -bubo*	-starts as a painless nodule the ULCER is painless, solitary, beefy red, granulomatous with elevated rolled edge bleeds easily -pseudobubo**	- <u>1st stage</u> starts as a transient vesiculopustule the ULCER is self-limited - <u>2nd stage</u> days-months after initial lesion constitutional manifestations Inguinal syndrome unilateral bubo Anorectal syndrome proctitis
Complications	-Phimosis -Phagedenic ulcers -Ulcerated bubos	-Urethral stricture -Rectovesical fistula -Genital elephantiasis	-Genital/anal ulcer, sinus, fistula -Urinary/rectal stricture -Genital elephantiasis -Malignancy
Lab	- Culture (selective medium)	- Giemsa stain of a piece of granulation tissue	- Culture (McCoy's medium) - Complement fixation - Microimmunofluorescence
Treatment	- Erythromycin , 7 days	- Cotrimoxazole , 14 days	- Doxycycline , 14 days

♠ herpes simplex progenitalis is the major cause of sexually transmitted genital ulcer in the developed countries.

* **Bubo**: inflammatory swelling of one or more lymph nodes in the groins.

** **Pseudobubo**: a subcutaneous granuloma, which may simulate inguinal lymphadenopathy

• What is the fate of prenatal (congenital) syphilis?

- ☐ Abortion
- ☐ Still-birth
- ☐ Early neonatal death
- ☐ Diseased-child:
 - **Early** congenital syphilis (simulating 2nd stage of acquired syphilis)
 - **Late** congenital syphilis (simulating late acquired syphilis)
- ☐ Free child

• **What are the manifestations of prenatal (congenital) syphilis?**

Early	Late
- Constitutional manifestations (marasmic features) - Rash - Bullous - Macules/Papules - Alopecia - Onychia - Mucous membrane lesions - Generalised lymphadenopathy - Other organ affection - Nephritis - Orchitis - Meningitis - Choroidoretinitis - Periostitis - Osteochondritis - Pneumonia	- Interstitial keratitis - Nerve deafness - Neurosyphilis - Bone affection - Joint affection "Clutton's joints"

• **What are the stigmata of congenital syphilis?**

Permanent features of previous syphilitic lesions, remaining for life.

Stigmata of Early syphilis	Stigmata of Late syphilis
- Saddle nose - High-arched palate - Bulldog face - Hutchinson's teeth - Moon's molars - Rhagades	- Corneal opacity - Optic atrophy - Sabre tibia - Frontal bossing - 8th nerve deafness - Perforated nasal septum/soft palate

• **How can you treat syphilis?**

	Drug of choice	Alternatives
Early acquired	BP 2.4 million IU IM x 1 dose	Doxycycline 100mg/12 hours x 14 days Tetracycline 500mg/6 hours x 14 days Erythromycin 500mg/6 hours x 14 days
Late acquired*	BP 2.4 million IU IM x 3 doses (1/week)	Same as above for 28 days
Neurosyphilis	AP 2–4 million IU/4 hours IV x 10–14 days	No alternative**
prenatal	AP 50.000 IU/ kg/ 8–12 hours IV x 10–14 days	

BP: benzathine penicillin

AP: aqueous penicillin G

* except for neurosyphilis

** patients allergic to penicillin should be desensitized

What is the Jarisch-Herxheimer's reaction?

Fever, chills, malaise, headache, exaggerated rash
Due to release of treponemal antigens after treatment

The patient should be followed-up at **1, 3, 6, 12 months** with quantitative VDRL.

Cure is considered when the result is negative or **4-fold** decrease is achieved.

VAGINAL DISCHARGE

What are the causes of vaginal discharge?

□ PHYSIOLOGICAL

- . puberty
- . premenstrual congestion
- . sexual excitation
- . pregnancy

□ PATHOLOGICAL

. vaginitis

most common

-infective

bacterial e.g. *G. vaginalis*

fungal e.g. *C. albicans*

protozoal e.g. *T. vaginalis*

-physical

e.g. foreign body

-chemical

e.g. antiseptics

. cervical

-infective

viral e.g. HSV, HPV

chlamydial e.g. *C. trachomatis*

bacterial e.g. *N. gonorrhea*, *T. pallidum*

-neoplastic

Why "vaginosis" NOT "vaginitis"?

It represents a disturbed vaginal microbial milieu, rather than a true infection

• What do you know about infective vaginitis?

	Candidal vaginitis	Trichomonal vaginitis	Bacterial vaginosis
Cause	- <i>C. albicans</i>	- <i>T. vaginalis</i>	- <i>G. vaginalis</i> - <i>M. hominis</i> - <i>Anaerobes</i> e.g. <i>Bacteroids</i> <i>Mobiluncus</i>
Presentation	-Severe vulval pruritus	-Vaginal discharge	-Vaginal discharge
Signs			
Vulvitis	++++	-+	-none
Vaginal discharge			
Amount	-minimal	-profuse	-scant/moderate
Odour	-sour	-offensive	-offensive, fishy
Consistency	-cheesy	-frothy	-homogenous, adherent
Lab			
Amine test*	- -ve	- -ve	- +ve
Microscopy**	- yeast (ovoid budding), hyphae	- protozoon (lagellated ovoid)	- clue cells♠
pH	-<4.5	->4.5	->4.5
Culture	-Sabouraud's	-modified Bushby	-blood agar
Treatment	-miconazole vaginal suppositories	-metronidazole oral tablets	-metronidazole oral tablets

*Addition of few drops of 10% KOH to a sample of vaginal discharge → immediate amine or fishy odour.

**Add one drop of normal saline to a discharge obtained by high vaginal swab.

♠Vaginal epithelial cells studded with Gram-variable coccobacilli.

AIDS

- **What is AIDS?**
Acquired Immuno Deficiency Syndrome.
- **What is the cause of AIDS manifestations?**
Defective CMI (Cell-Mediated Immune response)
- **What is the most important feature of such defect?**
Quantitative/qualitative defect of helper/inducer
CD4 T lymphocytes “the conductor of immunological orchestra”
- **What is its causative agent?**
HIV, Human Immunodeficiency Virus.
A retrovirus, first isolated in 1983 by Luc Montagnier and his colleagues.
- **How many patients have AIDS?**
About 2 millions, world-wide.
- **Where is the prevalence of HIV infection most rapidly increasing?**
South east Asia
- **Where is the greatest number of AIDS deaths?**
Subsaharan Africa.
- **What is the most common route of transmission?**
Blood-born.
- **Who is especially at risk?**
 - Homosexuals
 - Sexual partners of HIV-infected patients
 - Offspring of HIV-infected patients (transplacental/perinatal)
 - Drug abuse
 - Blood transfusion recipients
- **How long is the incubation period?**
Usually < 5 years.
- **Is it important to know the cutaneous manifestations of HIV infection?**
YES: Its prevalence is very high (85 – 100%)
It is often the first clue to the presence of AIDS.
- **What are the most common mucocutaneous features?**
 - Oral candidiasis
 - Seborrhoeic dermatitis
 - Xerosis
- **What are the most pathognomonic mucocutaneous findings?**
 - Kaposi's sarcoma
 - Oral hairy leukoplakia
 - Persistent, recurrent, treatment-resistant infections

What is the CDC?

Centers for Disease Control, Atlanta, Georgia

How do they classify HIV disease?

- 1- **Primary HIV infection**
Constitutional manifestations occur in some patients 3-6 weeks after infection
- 2- **Asymptomatic infection**
8-10 years
- 3- **Generalised lymphadenopathy**
2 or more extrainguinal sites
more than 1 cm
for more than 3 months
without explanation
- 4- **AIDS**
heralded by ARC (AIDS-Related Complex):
 - fever > 1 month
 - diarrhoea > 1 month
 - involuntary weight loss > 10%
 subgrouped to:
 - A-Constitutional disease
 - B-Neurological disease
 - C-Secondary infections
 - D-Secondary cancers

What are the mucocutaneous features of AIDS.

-Neoplastic

- Kaposi's sarcoma
- Non-Hodgkin's lymphoma

-Infectious

- Viral: herpes, CMV
- Bacterial: pyogenic, spirochaetal, mycobacterial
- Fungal: dermatophytes, pityrosporum, candidal, cryptococcal
- Protozoal: amoebic
- Arthropodes: scabitic

-Non-infectious

- Vascular: vasculitis, purpura
- Papulosquamous: psoriasis, seborrhoea
- Ichthyosis
- Oral: aphthosis
- Hair: premature graying, telogen effluvium
- Nail: color changes, deformities

- **What do you mean by “opportunistic infections”?**

Infection caused by endogenous or ubiquitous organisms of low virulence, occurring in debilitated compromised patients.

- **What is the most common bacterial pathogen in HIV infections?**

Staphylococcus aureus

- **How to use the lab to look for the virus?**

- Detecting the antiviral antibodies:

- **ELISA:**

sensitive (screening)

2nd generation has much lower false positives

- **Western blot:**

specific (confirmatory)

- Detecting the viral antigens:

- **p24 antigen**

- **gp120 antigen**

- Detecting the viral genome:

- **PCR**

- Detecting the Virus itself:

- **Culture** (the gold standard)

Is there more lab tests that can be done in such cases?

YES, non-specific tests can be of help e.g.

- CBC
- CD4, CD8 count
- STS
- β-microglobulin level
- Hepatitis-B virus
- Toxoplasma
- Tuberculin
- Chest x-ray

- **What is the goal of therapy?**

-Amelioration of symptoms in “full blown” AIDS

-Prevention of disease progression

-Treatment of secondary cancers and infections

- **How to achieve this goal?**

-Antiretroviral agents e.g.

Zidovudine

-Immunorestitution e.g.

Interferon

Interleukin

-Supportive measures

-Prophylaxis and treatment of cancers and infections

What is the mechanism of action of zidovudine?

A chain terminator of HIV reverse transcriptase

Does it eliminate the viral replication in circulating lymphocytes?

NO. It can only suppress such replication.

How could it be beneficial to the patient?

It improves the quality of life and survival rate

What is its most important side effects?

Bone marrow toxicity

بسم الله الرحمن الرحيم

ولا تقربوا الزنى انه كان فاحشة وساء سبيلا

الإسراء - الآية ٣٢

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BOXES

